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A PRACTICAL JOURNAL BUILT ON MERIT

EDITORIALS

IT'S WORTH SEEING

THIS is not written to boost the New York World's Fair. Many of us have been long since fed up on the publicity that seems to be a necessary part of such a gigantic venture. We ourselves, as a matter of fact, vowed that we would wait until October to visit the Fair, and then perhaps spend an hour or two there.

Somehow or other, we found ourselves there shortly after the opening—and one visit has led us there repeatedly since. The amusement areas, fireworks and water displays are impressive to be sure, but the exhibits of technical and educational value are even more to our liking. The mere creation of the fairyland of light and color upon the swamps and garbage dumps which formerly existed upon the site is a sufficient testimony to the power of man and a grandiloquent tribute to public health.

If you plan to go to the Fair, be sure to spend some time in the Medical and Public Health Exhibit. The building occupies a large ground space, is attractively and intelligently planned and architecturally pleasing. We thought it well done and in excellent taste, even before we learned that the architect was none other than the son of George Kosmak, nationally known physician and editor of *The American Journal of Obstetrics and Gynecology*.

The exhibit is, of course, planned and geared for lay consumption, but will prove interesting to any medico. We ourselves were particularly struck by the divisions on maternal welfare, cancer, tuberculosis and syphilis, but this probably reflects only the fact that these are fields of particular interest to us. The Popular Superstitions show is enthralling, and we lingered long at an exhibit describing cell division, just to hear high school and college students explaining to their less scientifically minded companions what was taking place.

Long lines of individuals are waiting at some exhibits. Over four million people have visited this building alone. These have undoubtedly come away with greater respect for scientific medicine than they had when they entered. It's marvelous propaganda—much needed in these troublesome and hectic days.

Best of all is the prospect that these carefully planned exhibits are not to be interred in rubbish heaps when the Fair closes, since the most valuable, together with other material, are to be perpetuated in an American Museum of Health to be established in New York City after the Fair itself is over. The Mayor of the city has promised to find a site and the money to support the permanent institute will be provided by philanthropic societies and insurance companies.

T. S. W.

NEXT TO VALOUR

IT is not the practice or the policy of THE AMERICAN JOURNAL OF SURGERY to comment on non-medical literature. However, we are deviating for once from the general rule to touch shortly on "Next to Valour," a novel by John Jennings, Jr., recently published by Macmillan. It is splendid writing, crammed with authentic and accurate history and enough adventure for any man.

We write this because the author is the son of John E. Jennings, member of our Editorial Board, although the novel can stand on its own merits. We were sorry when we had finished it—although it runs some eight hundred pages. T. S. W.

ALBERT MATHIEU

1881–1939

TO meet the deep and sudden sense of loss occasioned by the death of Albert Mathieu it is difficult to bring forward any of the factors which sometimes compensate. Mathieu was too young—if not perhaps in years, certainly in spirit—too productive, too vital, too strong in the hearts of those who were fond of him. Just as there seemed too little to prepare his friends for his death, there seems too little to compensate.

Albert Mathieu was born in 1881 in Faribault, Minnesota, where his early schooling was obtained. His medical studies were pursued at the University of Minnesota. After his required period of training he took up a general practice in South Bend, Washington, later moving to Raymond, Washington. During the World War he entered the army as a first lieutenant in the medical corps and served at Camp Lewis. His intimate contacts during the war with men well advanced in specialty practice convinced him of the ultimate futility of his former type of practice. At about the age of 37 he undertook a complete graduate training in the specialty of obstetrics and gynecology at the Lying-in Hospital in New York. In 1920 he began the practice of obstetrics and gynecology in Portland, Oregon, and registered continued progress in every branch of this specialty up to the actual moment of the illness which preceded his death.

Albert Mathieu was an independent and aggressive thinker. He was willing to stand behind a well-grounded opinion quite regardless of the opinions of others. He worked and studied constantly, wrote widely and well, held office in almost every important organization in his specialty and made for himself an enduring place in the hearts of his friends and intimates. He was a warm-blooded, sometimes impetuous man. He admired men of action and accomplishment, studied them and learned from them. Weakness, inconsistency, and lack of courage he despised. He was seldom if ever in robust health, especially in his later years. Yet he was always stimulating and amusing company to those whom he liked and understood. He was generous to a fault. Not everyone who knew him was his friend; yet he leaves unquestionably the deepest sense of loss and the most unmitigated sorrow.

ALBERT W. HOLMAN, M.D.

ORIGINAL ARTICLES

LAXATIVE INDUCED SPREADING PERITONITIS COMPLICATING ACUTE PERFORATIVE APPENDICITIS*

RESULTS OBTAINED WITH THE USE OF PERFRINGENS—CLOSTRIDIUM WELCHII ANTITOXIN AND A PARTIALLY MAINTAINED GLUCOSE METABOLIC BALANCE

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A preliminary report based on the treatment of twenty-five cases of ileus—fourteen with acute intestinal obstruction and eleven with spreading peritonitis—by the administration of perfringens (Clostridium welchii) antitoxin was published by Bower and Clark in 1928.[1] In nine of the eleven patients with spreading peritonitis, the causative factor was acute perforative appendicitis. A review of the literature in the United States prior to this date fails to reveal reports of the administration of perfringens antitoxin for the treatment of spreading peritonitis due to a perforated appendix.

In Europe, however, Delbet,[2] Micheal,[3] Williams[4] and Weinberg[5] had reported series of patients treated with the perfringens antitoxin. While the majority of patients treated had spreading peritonitis due to a perforated appendix, Delbet and Weinberg reported good results following its use in the treatment of the unruptured gangrenous appendix. I mention this because we have recently reported the finding of the antitoxin to the Clostridium welchii in the peritoneal exudate of patients operated upon for acute appendicitis in approximately 33 per cent of instances. The treatment of a nonperforative appendicitis by passive immunization or by drainage therefore is not indicated. In 1936, Priestly and McCormack[6] reported a series of thirty-two patients treated with perfringens antitoxin with good results.

Since the preliminary report in 1928, we have been using perfringens antitoxin in the treatment of peritonitis where anaerobes were suspected of playing a part in the etiology. Experimental investigations were conducted during the same period on several different methods of treatment; these and the results of a clinical study of spreading peritonitis have been reported.[7,8,9,10,11,12,13,14,15,16,17]

The main purpose of this report is to place before surgeons the immunologic aspect of the management of spreading peritonitis which had not been emphasized heretofore. While passive immunization may be considered the feature of this article, we realize that other factors, some of great importance, play a part in the outcome. These factors may be divided into two groups—those in which the surgeon has no part and those in which he plays an important rôle. In the brief abstracts which follow, reference will be made to a study

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of over 18,000 records of patients operated upon for acute appendicitis and peritonitis in twenty-eight hospitals in Philadelphia. Included in this group are 2,573 patients who had spreading peritonitis; their clinical records were reviewed a second time for a comparative study with the results obtained in this group of ninety-eight cases. The large group will be considered a control group and will be referred to subsequently as such.

Factors which influence the mortality of spreading peritonitis beyond the control of the surgeon are:

Age. Spreading peritonitis causes approximately 90 per cent of the deaths of patients admitted to hospitals with a diagnosis of acute appendicitis. The average age of those that die is 27 years; in the reported group 26 years.

Delay in Hospitalization. The average time between onset of symptoms and operation in the control group was 54.51 hours; in the reported group 93 hours.

Laxatives. The mortality increases with the administration of laxatives. In the control group a definite history of laxative administration was obtained in 1,408 cases; 153 did not receive laxatives, and sixteen, or 10 per cent, died; 1,119 had received one laxative, and 250, or 22 per cent, died; 135 had received more than one laxative, and 39, or 29 per cent, died.

In the reported group fifteen had not received a laxative, and one, or 7 per cent, died; fifty-six had received one laxative, and six, or 11 per cent, died; and seventeen had received more than one laxative, and two, or 12 per cent, died. The results obtained by the treatment outlined in this communication, I believe, are responsible for the great difference in the mortality percentage of the two groups. In this series of ninety-eight patients, eighty-eight, or 90 per cent, had received laxatives—this group is called, therefore, the Laxative Induced Spreading Peritonitis Group.

Complications. The most frequent complications of spreading peritonitis accompanying perforation of an acute appendix are acute inflammatory lesions of the lung and metastatic abscesses. The incidence of the complications in the control group cannot be used for comparison because of the small percentage of patients in the held group. In our own series the incidence of major complications in the immediate operative group was 21.15 per cent; in the held group 30.45 per cent. Metastatic abscess developed more frequently in the immediate operative group and pulmonary complications in the delayed.

Morphine Preoperatively. The percentage of patients suffering with spreading peritonitis who have been given morphine before admission to the hospital is gradually diminishing. That it still occurs too often, however, is shown by the fact that in one year fifty-five physicians in Philadelphia gave morphine to that number of patients before admission to the hospital and four died.

Enemas before Admission. Enemas may interfere with the localization of a spreading process; we have observed spreading peritonitis so induced postoperatively. Prior to hospitalization they are given infrequently as compared with laxatives, but when given, the result bears a direct relation to the stage and extent of the spreading process—given early in an advanced stage, the induced peristalsis may produce an increase in absorption sufficient to cause death; given late, when the process is regressing, a metastatic abscess only may develop.

Abdominal Palpation. Repeated abdominal palpation, particularly when forceful, interferes with the localization of a spreading peritonitis and should be avoided. Examination of the abdomen to determine the degree of rigidity and distention is necessary, but it can be done gently, the palm being placed so that contact with the epidermis only is made. Flexion of the distal phalanx will enable the examiner to estimate the degree of muscle tension. By applying the stethoscope—an instrument many surgeons have discarded—lightly to the abdomen at regular intervals, an ad-

vance or regression of the process can usually be determined.

Application of Ice Bag, Hot Water Bottle or Poultice. If patients would not use and the family physician would not order, but advise against applying a hot water bottle, ice bag or poultice to the abdomen in acute abdominal pain; if surgeons alone were given the prerogative of ordering them, the mortality of all the acute lesions of the abdomen requiring surgery would be tremendously diminished. What little good they may do is more than offset by the accompanying false sense of security. The patient may, by his attitude, influence the attending physician against operative interference.

FACTORS WHICH INFLUENCE THE MORTALITY OF SPREADING PERITONITIS CONTROLLED BY THE SURGEON

Correct Diagnosis. There are comparatively few errors of diagnosis made by surgeons when the serous membrane of the appendix is intact or when a frank spreading or local peritonitis is present, but the early subacute perforation with spreading peritonitis is frequently not diagnosed before operation. This is not difficult to explain. The gross difference between a localized mass, serosa intact appendix, which may be removed without contamination of the peritoneal cavity and an early perforation which cannot be removed without additional contamination of the peritoneal cavity is approximately 100 microns, yet if a spreading peritonitis develops following an attempt at removal of the latter the mortality is 360 times greater. Surgeons will do well to check over their diagnoses of early perforation; in this series the frank spreading peritonitis was diagnosed in 85 per cent of instances, the incipient perforation in 45 per cent.

The Evaluation of the Immunologic Response. Surgeons as a rule speak of this as the resistance or the reaction of a patient to an infection. An appreciation of the difficulties encountered in evaluating the immunologic response may be obtained by the following description of two cases which illustrate the two types most frequently observed by surgeons:

Case I. The acute inflammation of the appendix is accompanied by a high temperature and pulse, a marked increase in the neutrophiles, definite physical signs—this patient has an adequate immunologic response and is fortunate in two respects. The lesion is very likely to be diagnosed promptly and the patient operated upon before perforation occurs; if perforation does take place and he is properly managed, he will develop a high degree of immunity.

Case II manifests a milder immunologic response; moderate rise in temperature and pulse rate, little or no increase in neutrophiles —indefinite physical signs. This patient will react satisfactorily to the removal of the serosa intact appendix, but if there is perforation, his immunologic response will be commensurate with his preoperative symptoms and signs.

The immunologic response in the first case can be definitely evaluated by the surgeon; the patient's defensive mechanism has responded adequately to a virulent micro-organismal invasion. In the second instance, the surgeon cannot be so sure of his evaluation of his patient's reaction—it may be due to virulent micro-organisms operating in tissues of low resistance or the micro-organisms may be avirulent and the patient's response adequate. To summarize —these two cases, the prototypes of over 90 per cent of patients admitted with acute appendicitis, show that a consideration of the immunologic response is necessary only when the serosa is ruptured. In the unruptured, only one in 183 dies; in the ruptured, one in eight, or a difference of 2,200 per cent.

When to Operate. As previously stated, the immunologic aspect of the management is emphasized in this communication. It requires time for the development of either local or general immunity. This is just as true of spreading peritonitis as it is of a cellulitis, in the management of which surgeons have adopted the watchful wait-

ing principle. Gynecologists have applied this knowledge to the management of pelvic peritonitis.

Surgeons have been divided in their opinions regarding the value of time, the status of immediate or delayed operation, in the management of spreading peritonitis due to a perforated appendix mainly because immunologists have failed to submit conclusive evidence supporting either one or the other opinion. Recent investigations on the bacteriologic,[7] the immunologic,[8] and the clinical aspects,[9] of the problem seem to indicate that the factor, *Time*, is second in importance only to what is done at operation in the management of these patients.

Type of Anesthesia. The surgeon's responsibility to the patient includes the selection of the anesthetist as well as the kind of anesthetic to be administered. This is not difficult when operating for acute appendicitis. Patients with appendicial spreading peritonitis, however, frequently develop major complications and the type of anesthetic and its administration may be contributing factors in the development of the two most frequent, pneumonitis and metastatic abscesses.

Ether is still the safest general anesthetic, but Mengle[18] has shown that in the earliest stages of a spreading peritonitis, the respiratory excursions accompanying its administration increase the rapidity of peritoneal absorption. In addition, the movements of the diaphragm, the diminished relaxation of the abdominal muscles and the frequency with which distended loops of intestine crowd into the operative field, make the locating and quarantining of the infected area more difficult. This entails more time hence more anesthetic; these, together with the trauma to the endothelial cells because of the increased manipulation, result in an increase of absorption of toxic products which cannot always be successfully combated by the patient.

Local novocaine was used most frequently in this series because the initial operation in the majority of patients consisted of an incision and the insertion of a single drain. Spinal anesthesia was the second choice. The relaxation of the abdominal muscles which it alone produces, facilitates exposure of the process and apparently diminishes toxemia.

An analysis of the major complications and their relation to the kind of anesthetic administered in this group shows that spinal anesthesia in all probability did not contribute materially to the reduction of the incidence of complications involving the respiratory system. However, this small series of cases does not justify a definite statement. In my opinion if, when spinal anesthesia is used, its effect can be kept below the level of the sixth rib, it is the anesthetic of choice; the one exception being a well localized appendicial abscess which can be operated upon under local anesthesia. When the anesthesia ascends to the level of the fourth rib, the intercostal muscles are paralyzed, the excursion of the diaphragm may be interfered with, a drop in blood pressure occurs, blood stagnates in the right chest and passive pulmonary congestion occurs, presenting a condition favorable to the development of a pneumonitis.

What effect does the deferring of the operation have on the development of postoperative pulmonary complications? Of the eleven patients who died, four developed a lobar pneumonia; in two this disease was given as the cause and in two others the contributory cause of death; two were operated upon immediately; two were held; three of the four were given spinal anesthesia and one, ether.

What Is Done at Operation. Simple drainage, the search for or the removal of the appendix; the procedure depends to a great extent upon what the surgeon's past experiences have been in the management of these cases. Patients with spreading peritonitis are operated upon by two types of surgeons—those who operate routinely on all cases and those who consider the operation only a part of the management

of this condition. Scientific management is based on dose of antigen and the reactive capacity of the patient, which for the present can best be determined by studying closely the gross changes at operation and correlating them with symptoms and signs before, and what happens to the patient after operation.

What Is Done if the Patient Is Not Operated upon Immediately. The importance of a preoperative and postoperative management instituted and carried out by a group composed of the chief surgeon, associates, intern and a competent clinical pathologist who is capable of making and evaluating Schilling hemograms, cannot be overestimated. A disease which carries with it a mortality of from 25 to 40 per cent, requires constant medical supervision. It is not a one man job—a patient's chance for recovery is increased markedly with such a group in attendance. The treatment should consist of Fowler position, nothing by mouth, perfringens antitoxin or convalescent serum or both intramuscularly, glucose and saline intravenously or by hypoclysis, morphine as indicated, and mental and physical quietude, which are always indicated.

What Is Done if the Patient Has Been Operated upon Immediately. Same as above except that abdominal dressings are not changed unless absolutely necessary and drains are not disturbed usually until the seventh day.

THE ADMINISTRATION OF PERFRINGENS ANTITOXIN IN THE TREATMENT OF SPREADING PERITONITIS DUE TO A PERFORATED APPENDIX

Perfringens antitoxin was first administered in the United States in the treatment of spreading peritonitis in 1927;[1] in 1928, we began our experimental investigations as has been stated. In one of our reports, we outlined the results of the treatment of a large number of dogs in which spreading peritonitis had been induced by ligating the appendix and its mesentery and giving large doses of castor oil.[10] Our conclusions from this experimental work were responsible for a modification of the method of administration, dose, etc.

The perfringens* used in the treatment of the series of patients previously reported and in our early experimental work was the unconcentrated serum; 50 c.c. contained 10,000 units of perfringens antitoxin. During the past two years, we have used the concentrated serum, the 20 c.c. syringe containing 10,000 units each of perfringens and vibrion septique antitoxin with an excess estimated to be 30 per cent or over.

Indication for Treatment. Patients with serosa intact appendices and those with localized abscess were not given the antitoxin; only those with spreading processes following perforation were treated. They were divided into two groups; those operated upon immediately and then given perfringens and those held, given perfringens and then operated upon. The condition of patients on admission determined the group in which they were placed—the very sick were usually held; those in which the diagnosis was uncertain in most instances were operated upon.

Method of Administration. Patients were always tested for sensitivity. In the initial series, the antitoxin was given intravenously. When we discovered that better results were obtained by using the intramuscular methods in dogs, we used it in the treatment of man. We prefer to inject the antitoxin 2 of 3 cm. below the crest of the ilium, slightly posterior to the midlateral line. It is advisable to select a site for injection away from the lumbar region, especially on the right side; otherwise the swelling and tenderness which may follow might make it difficult for the examiner to differentiate between tension or rigidity due to tissue reaction and that due to an intraperitoneal process. Because of the frequency with which the appendix is located retrocecally, abscesses in this region are not uncommon.

* Supplied by the Research Laboratories of Sharp and Dohme.

Time of Administration. Patients who were held were given the antitoxin as soon as the diagnosis was made. Those operated upon received their injection immediately upon return from the operation.

Dose. While the maximum result—lowest mortality rate in dogs—was obtained where the total amount of antitoxin given averaged 3 c.c. per kilo of body weight, it will be observed that this amount was infrequently given in the cases reported. In this series, the average dose given intramuscularly was less than 1 c.c. per kilo of body weight; the average dose per patient, whether operated upon or held, was 44.82 c.c.

Perfringens Antitoxin Combined with Whole Blood and Convalescent Serum. When we had reached what appeared to be the minimum mortality with the use of perfringens antitoxin alone, in our experimental work, we tried combining it with whole blood, but the results were approximately the same. By using convalescent serum alone, however, taken from dogs that had recovered after spreading peritonitis, we reduced the mortality below the minimum obtained by the administration of perfringens antitoxin.[10]

We then tested lyophile convalescent serum of both man and dog after it had stood as long as 444 days after lyophilization and found in both instances that it had maintained its original antitoxic titer. We have, therefore, used both fresh and lyophilized convalescent serum alone and in conjunction with perfringens antitoxin with excellent results. When the spreading process develops in a patient with low resistance as shown by a leucopenia and a marked shift of the Schilling index, we use transfusion of a small amount of citrated blood in conjunction with perfringens antitoxin or convalescent serum.

The Effect of Perfringens Antitoxin on the Toxemia of Spreading Peritonitis. None of the forty-six patients who received perfringens antitoxin as a part of the treatment for localization before operation developed delirium. However, it occurred in one instance postoperatively, in a patient who on admission had a urea nitrogen of 50 and developed uremia. In those who were operated upon and then given the antitoxin, mental irritability and delirium were not uncommon. One patient, became delirious on the fifth postoperative day, got out of bed to calm a restless fellow patient and contracted pneumonia. I feel that this catastrophe could have been averted had the patient had a sufficient amount of antitoxin. This statement is substantiated by the following experiences:

A spreading peritonitis in a male, age 44, developed following an acute perforation of a diverticulum of the sigmoid. This was localized, a colostomy was done and an abscess drained. Ochsner treatment, including intravenous glucose solution, was instituted after operation but the patient developed a delirium which persisted for three days. Anaerobic cultures of the purulent exudate showed Clostridium welchii. Following the administration of perfringens antitoxin, delirium immediately subsided.

A second patient, a female, age 56, following a Kraske operation for carcinoma of the rectum, developed an infection of the operative field with an associated toxic psychosis. She failed to respond to the intravenous injection of glucose but improved immediately after the injection of perfringens antitoxin. Clostridium welchii was isolated culturally from the purulent discharge.

Because of the high incidence of a polymicrobic flora in spreading peritonitis, the variability of the patient's immunologic response and the parenteral administration of glucose, one could not definitely ascribe the usual drop in temperature and pulse which accompanies the localization of a spreading process solely to the administration of perfringens antitoxin.

Nausea and vomiting were rare in those given perfringens before operation but not infrequent in those operated upon immediately.

The Effect of Perfringens Antitoxin on Physical Signs. Except in the frank, fulminating type of spreading peritonitis, an examination of the abdomen may not

present sufficient evidence to enable the surgeon to diagnose a spreading process definitely. The most important physical sign is *rigidity*, but even this may be absent, or it may be difficult to evaluate the degree. This is especially true in young children and in neurotic and obese individuals. When rigidity is present before the administration of perfringens antitoxin we have noted an improvement, usually beginning in the upper left quadrant, twenty-four hours after the injection. Rigidity is gradually replaced by increase in tension, followed by relaxation, which reaches its maximum about the seventh day of the disease. A differentiation between voluntary spasm and actual rigidity can best be determined by rectal examination. We believe that this should be done only at the initial examination of the patient and again directly preceding the operation.

At the second examination one may be better able to outline the process by having the patient assume the right lateral prone position with the knees slightly flexed. Free fluid which was present on admission is usually completely absorbed forty-eight hours after the first injection of prefringens antitoxin. Distention, if present, gradually lessens, liver dulness usually returning to normal between the fourth and fifth days of the disease. We have only rarely been concerned about treatment for distention; Wangensteen siphonage was used in less than 5 per cent of patients who were held before operation in this series.

The Effect of Perfringens Antitoxin on Peristalsis. Peristalsis is actually a physical sign, but its importance in the diagnosis and prognosis of the disease justifies a separate discussion. It is not always entirely absent in a laxative-induced spreading peritonitis secondary to a ruptured appendix. We occasionally hear sounds of moderate degree in the left lower and upper quadrants and faint tinkles in the right lower abdomen. While its presence may be confusing, its absence is almost always significant of a spreading process. Peristaltic sounds heard immediately or within a few minutes of the swallowing of fluid may at times be misleading. The duodenum, jejunum and upper ileum may contract normally, the sounds being heard clearly in the upper right abdomen immediately after taking fluid, when localization of a spreading process is occurring in the right lower quadrant. Peristaltic sounds are seldom heard in the localization region, however.

When peristalsis has been absent and perfringens antitoxin has been administered, we have consistently heard the first sounds presaging the return of normal intestinal contraction in the upper left quadrant twenty-four hours after the injection. Within twelve to twenty-four hours thereafter, there is a return of peristaltic sounds in the lower left or upper right abdomen, depending in which direction the initial spread of the infection was greatest. It is difficult at times, particularly in children, to be sure that peristalsis is not present and it is more difficult to interpret any sounds that may be heard. The initial contractions of the ileum or perhaps the jejunum, may be likened to a tinkle. As the intestinal tone returns, the sound develops more volume and frequently culminates in a miniature explosion.

We are convinced that perfringens antitoxin promotes an early return of intestinal tone. We have repeatedly observed patients who, following its administration, have had bowel movements on the third day, and frequently diarrhea. In this series, 38 per cent of the patients had a spontaneous bowel movement on an average of eighty-four hours after the first injection of perfringens antitoxin. Diarrhea was present in four cases. It may be that the reaction of the mucosa of the intestine to the antitoxin is similar to the reaction of the epidermis when an urticaria develops; the resulting congestive edema involving the mucosa may stimulate peristalsis and cause the spontaneous evacuation.

An Attempt to Maintain a Glucose Metabolic Balance. The ninety-eight patients in this group were admitted to the

hospital on an average of ninety-three hours after onset of symptoms. Each patient was restless and irritable unless he had been given morphine; the temperature and pulse had increased but had not reached a maximum; the tongue and skin were dry and the abdomen was moderately distended. During the ninety-three hours preceding admission, the patient could not retain fluids. He was seriously ill but was in what might be called "the compensatory stage," "compensating" because the liver was supplying the circulating cells with nourishment in the form of glycogen. How long can the liver do this adequately? When does the "decompensation" stage begin? There is no recognized laboratory test that can determine this. There are no definite premonitory symptoms and signs.

There is, however, one function of the liver, which if utilized to the utmost during the compensatory stage, will avert disaster. It has been proved experimentally that the liver of both man and dog can metabolize approximately 1 Gm. of glucose per kilo of body weight per hour. The average weight of the patients operated upon in this series was 55 kilos. The normal liver, therefore, could dispose of 1,320 Gm. of glucose in twenty-four hours, the equivalent of 5,280 calories. If the average liver can supply the body with this number of heat units, it is reasonable to suppose that there are times when it may be called upon to do so.

In the "compensatory" stage of spreading peritonitis, therefore the temperature is elevated; metabolism is increased; consumption of glycogen is increased over the normal. How can we supply the liver with this necessary amount of fuel? *A continuous intravenous injection* of 5 per cent glucose solution, administered thirty drops to the minute, will supply 2,700 c.c. of fluid in twenty-four hours. This amount of fluid is sufficient to maintain water balance but will supply only 135 Gm. of glucose, which when metabolized, amounts to approximately 540 calories. An attempt should be made to maintain metabolic as well as water balance.

If a patient in the "immunity developing stage" of spreading peritonitis reacts satisfactorily when given $\frac{1}{10}$ of *what the liver can dispose of in the form of glucose, then he should react very much more satisfactorily with* $\frac{1}{5}$ *to* $\frac{1}{3}$ *of what the liver can dispose of.* A continuous intravenous injection of 10 per cent glucose solution, administered thirty drops to the minute, will supply a patient with 270 grams of glucose in twenty-four hours or the equivalent of 1,080 calories. We use this routinely during the early stages of the disease, diminishing the percentage of glucose when sugar appears in the urine.

SUMMARY AND CONCLUSIONS

1. The results of the management of ninety-eight patients in whom spreading peritonitis developed as a complication of acute perforative appendicitis are presented.

2. Ninety per cent of these patients received one or more laxatives. For this reason, the term "laxative-induced spreading peritonitis" is used.

3. Laxative-induced spreading peritonitis is more virulent than spreading peritonitis not so induced. Laxatives, their dose, number and kind, influence mortality.

4. The factors influencing the mortality of a spreading peritonitis are discussed in order of their importance.

5. The results of the administration of perfringens antitoxin in the treatment of laxative-induced spreading peritonitis are outlined.

6. The importance of a more complete glucose metabolic balance in the treatment of laxative-induced spreading peritonitis is emphasized.

7. The results in a group of patients suffering from spreading peritonitis who were operated upon immediately are compared with those in whom operation was deferred.

Two tables are appended showing the comparative results obtained in the management of patients with spreading peritonitis due to a perforated appendix by

operating immediately and by delaying operation until a local and a general immunity had developed.

The management of the two groups was identical with the single exception of the time at which operation was performed.

TABLE I

LAXATIVE INDUCED SPREADING PERITONITIS COMPLICATING ACUTE PERFORATIVE APPENDICITIS

Treatment—Perfringens Antitoxin—Glucose Metabolic Balance

	No. of Cases	No. of Deaths	Average Age	Time between Onset and Operation (Hours)	Average Dose of Perfringens, C.C.	Per Cent Mortality
Immediate operation..	52	8	27	54.60	44.73	15.30
Delayed operation	46	3	22.11	196.10	44.91	6.52
	98	11				

TABLE II

LAXATIVE INDUCED SPREADING PERITONITIS COMPLICATING ACUTE PERFORATIVE APPENDICITIS

	No. of Cases (98)	Appendix Removed			Complications	
		No. of Cases	No. of Deaths	Per Cent Mortality	Incidence of Major Complications	Per Cent Mortality
Immediate operation.....	52	8	3	37.5	21.15	15.39
Delayed operation	46	2	0	0	30.43	6.52

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ABDOMINAL AUSCULTATION*

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THE literature of abdominal auscultation is relatively barren. We note auscultation referred to occasionally in articles dealing with abdominal problems, but its importance is stressed mainly in the English literature. No complete, concise report has appeared recently which emphasizes its value, interpretation and technique. The information it yields warrants careful study by internist and surgeon, especially in the differential diagnosis of the acute abdomen where it plays its most important rôle. We wish to record some clinical information which the stethoscope has yielded and advocate its addition to the armamentarium of the abdominal diagnostician. It is not our intention to explain the mode of production of every single sound. Such work is in progress at the present time in physiologic laboratories. It is well to recall, in this connection, the excellent work done by Dr. Charles B. Puestow on intestinal motility. This essay, however, deals solely with the auscultatory phenomena.

To understand fully and to interpret the findings we must briefly review the accepted physiology of the gastrointestinal tract. The term "peristalsis" is used in a general way to designate the wave of muscular contraction in any hollow viscus. In a restricted sense the term may apply to an intestinal movement which consists of a wave or muscular contraction preceded by a wave of relaxation. We refer to this progressive phenomenon by such terms as "the law of the intestine" or "myenteric reflex." Cannon has subdivided peristaltic movements into the following:

(*a*) *Diastalsis:* a downward wave of contraction preceded by a wave of inhibition which is manifested chiefly by the small intestine. Sometimes these waves travel rapidly for a length of 2 or 3 feet and we then refer to them as "peristaltic rushes."

(*b*) *Catastalsis:* a downward wave of contraction *not* preceded by a wave of inhibition and manifested chiefly by the stomach.

(*c*) *Anastalsis:* an upward moving wave of contraction not preceded by a wave of inhibition (reverse or antiperistalsis). This reverse action is normally manifested by the proximal colon but may also appear as an abnormal manifestation or any portion of the alimentary tract.

There are some movements of the intestinal tract that are not strictly peristaltic in nature. For our purpose, we shall mention a few of these:

Rhythmic segmentation—a localized rhythmical contraction found in the small intestine which churns and subdivides the masses of food within the intestinal lumen.

Pendulum movements—which manifest themselves by a to and fro movement of the small intestine.

Haustral movements—local contractions of the colonic haustra, analogous to the rhythmic segmentation of the small intestine.

Carey has made some interesting observations on the anatomy, and histology of the bowel musculature. He disagrees with the current concept that the muscle wall consists of an inner circular and an outer longitudinal layer. By interesting experiments, he rather conclusively shows

* Read before the Chicago Surgical Society, November 5, 1937.

that instead of an inner circular layer there is really a closely coiled oblique layer. He further shows that instead of an outer longitudinal layer of muscle we are dealing with another oblique layer, the coiling of which is more elongated. He further states the inner layer makes one complete turn around the intestinal lumen within a distance of every 5 mm. while it takes 15 mm. for the outer layer to make one complete turn.

Visualization of the result of the contraction of these two spiral muscles, makes it logical that the cephalad constriction is caused by a contraction of the inner layer while the caudal dilatation is produced by a contraction of the outer oblique layer. We believe that the sounds produced are a combination of two factors, (1) the contracting muscle causing (2) the onward passage of intestinal contents within the lumen of the gut.

As to innervation, peristalsis is supposedly under the control of the extrinsic and intrinsic nerves. The extrinsic control is via the sympathetic (splanchnic) nerves which inhibit, and the parasympathetic (vagus) fibers which augment peristaltic action. The intrinsic mechanism is provided by the Meissner and Auerbach plexuses.

When one listens to an abdomen for the first time he may be baffled by the number and types of sounds heard, but such variations are to be expected in view of the varieties of peristaltic movements. With a little practice normal sounds can readily be differentiated from the abnormal, not only as to quality but also as to intensity and frequency. To describe sound in words is difficult, so that we must use rather commonplace descriptive terms and convey our auditory perceptions by comparing them with analogous sounds from other sources.

Normal peristaltic sounds may be described as resembling gurgles, rustlings or clicks which tend to be continuous or even overlapping. They are usually more continuous than the heart sounds and relatively homogeneous. When slowed to the respiratory rate they are said to be definitely diminished and, when several respirations pass without a single peristaltic sound being heard, they are considered greatly diminished. They are normally not heard without the use of the stethoscope. Conditions which slow peristalsis tend to make the sounds fainter; louder sounds usually accompany an increased peristaltic rate. The exceptions to this rule will be discussed subsequently.

Normal variations occur in both rate and tonal intensity. The intake of food, drink and certain drugs stimulates peristalsis and increases sounds. Starvation, repeated vomiting and depressant drugs have the reverse action. Among such drugs are ether, chloroform and alcohol.

Air-swallowing and excessive colonic irritation may produce booming or explosive noises audible without a stethoscope and even at some distance; but they are not ringing or resonant like obstructional borborygmi.

Sound variations in pathologic conditions group themselves into three similar categories, namely, diminished, increased and abnormal sounds.

Complete intestinal paralysis produces complete peristaltic silence. The late Dr. J. B. Murphy used to refer to the abdomen in acute diffuse peritonitis as being "silent as the grave." Perforated peptic ulcer, diffuse appendicial peritonitis, abdominal gunshot wounds, acute pancreatitis and various acute peritonitides belong in this category. Large abdominal acute effusions of blood, bile and urine produce peristaltic silence; smaller ones at least a relative quiet.

In intestinal paresis, i.e., incomplete paralysis, the abdomen is quiet but not silent; e.g. in lesser effusions of blood, bile and urine, in the serous peritonitis or "wet belly" or acute appendicitis preceding perforation; in the acute serous or serohemorrhagic effusion of twisted pedicles, in the lesser hemorrhages of ruptured ectopic pregnancy, bleeding corpus hemorrhagicum or corpus luteum and abdominal injuries with some hemorrhage but no peritonitis.

A ruptured Graafian follicle diminishes peristalsis only mildly as a rule.

After general anesthesia the peristaltic sounds gradually return to something like normal in from twenty-four to forty-eight hours, the time of recovery being further prolonged by postoperative atony, abdominal hemorrhage and, most of all, by peritonitis. Neisserian peritonitis produces more often a quiet than a silent abdomen.

The peristaltic sounds are increased in incomplete mechanical ileus, spinal anesthesia, active catharsis and intraintestinal hemorrhage, such as in bleeding peptic ulcer and, probably, ruptured esophageal varix. In incomplete mechanical ileus the increased sounds are due to hypertrophied musculature of the bowel wall. In spinal anesthesia the sympathetic nerves are believed to be anesthetized, permitting the parasympathetic augmenters to act unopposed. In intraintestinal hemorrhage, blood within the intestinal lumen stimulates peristalsis in the same manner as food. In bleeding peptic ulcer the stethoscope becomes an important diagnostic aid in differentiating intraintestinal bleeding from intraperitoneal bleeding. In the former peristalsis is increased; in the latter peristalsis is greatly diminished or absent.

Obstructional borborygmi are peristaltic sounds, metallic and tinkling, bubbling or loud and resonating in character, and are pathognomonic of incomplete mechanical ileus. They are heard in no other condition. The metallic tinkling borborygmi must be differentiated from similar, metallic, splashing sounds in complete ileus which are passively produced by the palpating hand, stethoscope or diaphragmatic movement. The resonating or ringing borborygmi are produced by the peristaltic movement of distended intestinal segment lying proximal to the seat of obstruction and containing both gas and. liquid Normally, there is little or no gas in the small intestine. The presence of this metallic tinkling type of sound, therefore, signifies some variety of ileus of relatively high degree.

Among other abdominal sounds are: transmitted cardiac and respiratory sounds, splenic and liver friction rubs, the splashing sounds of pneumohydroperitoneum, coin sound, the bruit of abdominal aneurysm, crepitation from subcutaneous emphysema and the so-called mythical (?) "bruit de collision" of large gallstones knocking against one another.

Cardiac and respiratory sounds are not transmitted through the normal abdomen, but they are heard throughout the abdomen in intestinal distension. In the otherwise silent abdomen of complete paralytic ileus, the cardiac and respiratory sounds are usually strikingly prominent, being the only sounds heard except for the occasional adventitious splashes or tinkles previously mentioned.

A splenic or hepatic rub is usually due to the presence of fibroplastic exudate between visceral and parietal peritoneum and indicates a fibrinous peritonitis. A pleuritic rub is ruled out by noting the absence of breath sounds.

In a case of phlegmonous gastritis a to and fro friction rub was heard just above and to the left of the navel (abdomen shaved) where the stomach comes in contact with the anterior abdominal wall.

The splashing sound of pneumohydroperitoneum has not been described previously so far as we can determine. We have heard it only once in a case of perforated carcinoma of the stomach which showed a large pneumoperitoneum and a long fluid level in the free peritoneal cavity. We heard it first when the patient hiccuped. The sign may be elicited too, by having the patient inhale deeply and then forcefully and rapidly exhale. It is a characteristic tinkling splash like that in hydropneumothorax.

Coin sound in pneumoperitoneum is transmitted as characteristically as in pneumothorax and more loudly than in tympanites. Together with auscultatory percussion and the rubbing or scratching sound tests, this can outline the gas bubble somewhat more accurately than percussion alone.

In ascites the transmission of a fluid wave on percussion is accompanied by a "double tap" sound which is not generally known. The "double tap" is not transmitted by a fat abdominal wall as is the percussion wave at times. The tone character of the "double tap" is about like that of the so-called "split second sound" of the heart. The two taps come very close together and are due to the fact that the percussion sound wave travels through ascites a little faster than the fluid wave. Two other auscultatory tests—the "scratch and rub tests" produce a sound which is easily transmitted through meteorism and pneumoperitoneum but not through solid or cystic tumor, fluid or through the normal abdomen unless very slender.

To illustrate, we shall describe the sounds in some typical abdominal lesions. In unruptured acute appendicitis, during the first twenty-four to thirty-six hours, peristalsis is usually normal or even mildly increased; but as a serous peritoneal effusion ("serous peritonitis" or "wet belly") appears, the sounds diminish in strength and frequency and, "the wetter, the quieter." When perforating, purulent spreading peritonitis sets in, peristalsis rapidly disappears, the abdomen becomes silent (paralytic ileus) and tympanites develops in the silent, tender belly. As the appendicial inflammation becomes walled off and an inflammatory mass or "abscess" is formed, mechanical ileus due to adhesions, kinks, and inflammatory edema of the small bowel may result. In the lesser degrees of obstruction, there are resonant bubblings and gurglings in "rushes" and in the higher degrees, metallic tinklings and clicks with long silent intervals. If the abscess disappears or is evacuated, the sounds gradually return to normal as the intestines free themselves.

After a celiotomy, the abdomen is usually silent (traumatic paresis?). Intestinal sounds normally begin to return within the first twenty-four to forty-eight hours after operation. If the abdomen remains silent longer, we may suspect some intraperitoneal complication such as peritonitis or hemorrhage.

Infections of the abdominal wall do not alter peristaltic sounds except by spreading to the peritoneal cavity.

Perforated peptic ulcer presents probably immediately (reflex?) a silent abdomen of the quietest type. In the forme fruste type of ulcer perforation, peristaltic sounds begin to return soon after leakage stops and they strengthen as peritonitis subsides.

The sounds are loudest with chronic incomplete mechanical ileus. Here the bowel may become excessively hypertrophied and the sounds so loud as to be audible without the stethoscope. As the obstruction becomes more complete, the "rushes" and other sounds become fewer and metallic "tinklings" make their appearances until they too become blotted out in the silence of complete obstruction; in brief, "the tighter the obstruction, the quieter, the abdomen." The silent obstruction brooks no delay, if it is not already seen too late. The noisy obstruction may have a food-obturation factor removable by oil and flushings. It can also be a gangrenous Richter hernia.

Acute salpingitis produces the least silent acute perforative peritonitis, perhaps because the sigmoid, ileum and omentum wall off the Neisserian infection in the pelvis and prevent its upward spread, so that most of the intestine continues to function and produce sounds. Here, too, kinking of the ileum may result in mechanical obstruction and obstructive borborygmi be the guide to its diagnosis.

In extra-abdominal lesions, such as pericarditis with effusion, coronary thrombosis, angina pectoris, pneumonia, dissecting aneurysms, diaphragmatic pleurisy and acute osteomyelitis, the peristaltic sounds remain normal unless diminished by fever, abstinence from food or other accompanying conditions. Neither does renal colic, pyelitis, acute unruptured cholecystitis materially affect peristaltic sounds per se.

In the traumatic abdomen the persistence or early return of normal sounds is a

rather certain reassurance of absence of serious intraperitoneal damage. On the other hand, should the abdomen become silent and remain so, we have an indication for surgical intervention unless pain, tenderness and rigidity are absent.

In acute rupture of the bladder, if the urine is sterile, the sounds gradually disappear, while with infected urine, they disappear rapidly.

The abdomen should be auscultated both before and after attempting reduction of a hernia. If a viable segment of intestine is reduced the obstruction is completely overcome, the sounds reappear and become increasingly normal and vigorous. If obstructive borborygmi were present before reduction they gradually return to normal tonality following reduction. If a hernia is reduced without relieving the obstruction (reduction *en bloc* or of a gangrenous loop) the abdomen remains silent in incomplete obstruction, or borborygmi appear if obstruction remains incomplete. When an intestinal perforation follows taxis, the abdomen becomes silent instead of regaining normal sounds and becomes increasingly tender, instead of less tender, until it reaches the absolute silent stage of a well-developed peritonitis.

CONCLUSIONS

1. Abdominal auscultation deserves a place in the diagnostic aramentarium of the physician.

2. Normal and abnormal sounds have been discussed and described.

3. The characteristic silence of spreading peritonitis is emphasized.

4. The significance of obstructive borborygmi is outlined.

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THE OPERATIVE TREATMENT OF SCOLIOSIS*

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THE objective in the treatment of scoliosis should be completely to correct the curvatures and maintain correction. That many curvatures can be completely corrected cannot be denied; indeed, with some of the mild ones, or even the moderately severe ones, lateral bending alone is sufficient to do this. Others with severe deformity and accompanying trunk displacement cannot be so corrected, but they are by no means beyond help. With treatment in these cases the deformity may be lessened and the trunk displacement reduced. The problem is how to maintain the correction or the correction and compensation. This we believe is best done by operative fusion of a portion of the spine.

All patients who are known to have had poliomyelitis as well as those who have curvatures, are referred to a special division of the clinic. Such patients are given a general physical check-up. Sitting and standing heights are measured, as well as the length of the lower extremities. At the same time the list or displacement of the trunk is estimated. Then roentgenograms of the entire spine are taken both in the sitting and recumbent positions. At times one needs further roentgenographic study, which may be done later. The patient is sent to the gymnasium for exercises to improve posture, increase flexibility of the spine, and keep him in close touch with the clinic. This is often continued because of delay in securing operative permission.

On the data obtained we should be able to give a prognosis. Risser and others have observed that a curvature will increase until the growth period has ceased, and after that there will be no further increase in the deformity. This has been found to be true for all curves, regardless of etiology. At this point one must select the proper patients for treatment by fusion. Cases selected must warrant the long time spent in the hospital that is necessary for a satisfactory result.

The next problem is to estimate what result may reasonably be expected with correction, or correction and compensation, followed by operative fusion of the spine.

The patients that are suitable candidates for such treatment may be divided into the following groups:

1. Those with mild or very flexible, progressing curves that may be completely corrected. (Figs. 1–4.)

2. Those with moderate curves where partial correction is sought, as well as the reëstablishment of body balance. (Figs. 5–8.)

3. Those with severe deformity, together with marked displacement of the trunk, where appreciable correction of the primary curve is not the sole object, but rather the reëstablishment of body balance. (Figs. 9–11.)

4. Those with undue pain or fatigue, regardless of the mildness of severity of the deformity, even if the growth period has passed.

The patients in the first group are advised this treatment especially when further growth is expected because we know that to let such a curve go untreated would result in the development of structural changes in the thorax and vertebral bodies, and as time goes on the possibility of complete correction would be proportionately lessened. On the other hand, to advise such treatment on a young or immature curve where the ends are not definite and the

* Excerpts from thesis presented at Columbia University, June, 1936 as partial fulfillment for the requirements for the degree of Doctor of Medical Science. This work was done at the New York Orthopedic Dispensary and Hospital, New York City, and is based on an end-result study of 255 patients operated upon in the years 1926–1935 inclusive.

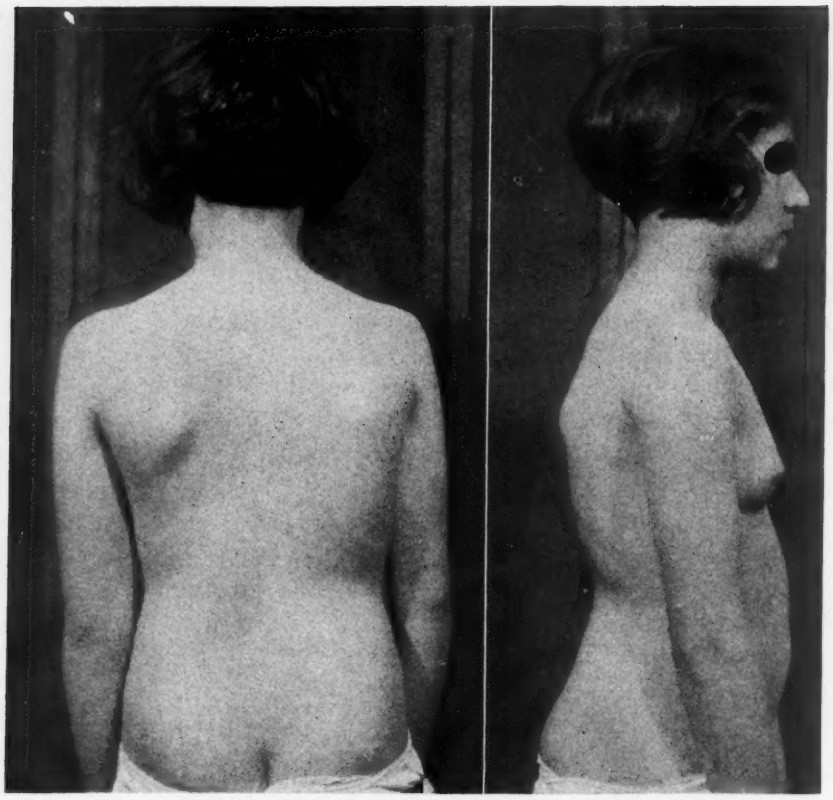


Fig. 1. Fig. 2.

Figs. 1 and 2. Back and side views of patient in Group 1.

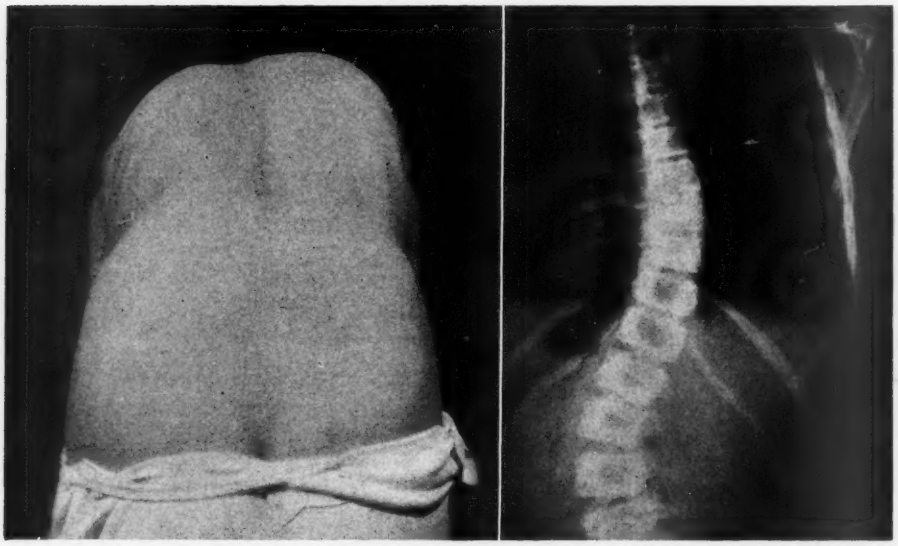


Fig. 3. Fig. 4.

Figs. 3 and 4. Forward bending and *x*-ray views of same patient as in Figures 1 and 2.

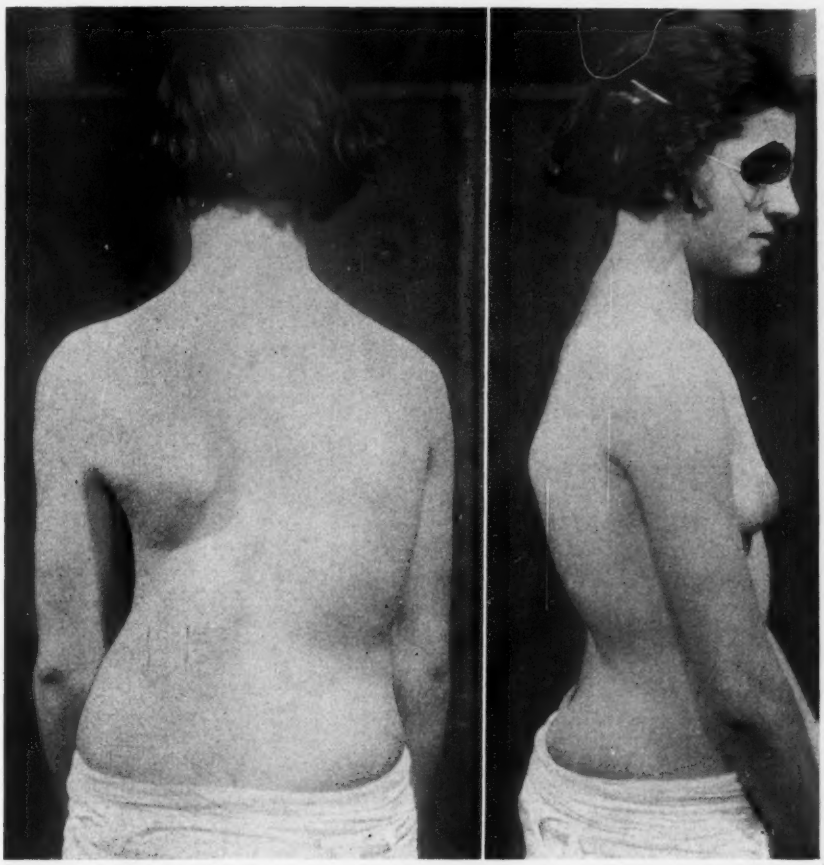


Fig. 5. Fig. 6.

Figs. 5 and 6. Back and side views of patient in Group 2.

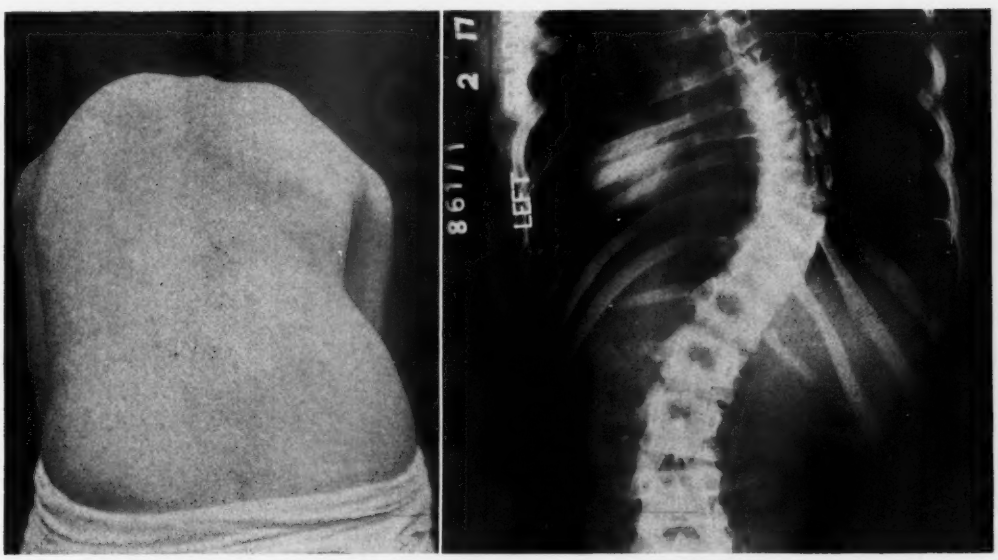


Fig. 7. Fig. 8.

Figs. 7 and 8. Forward bending and *x*-ray views of same patient as in Figures 5 and 6.

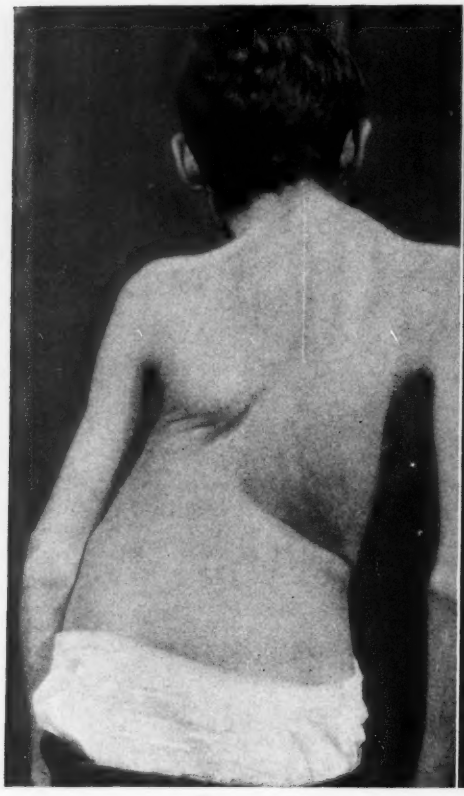


FIG. 9.

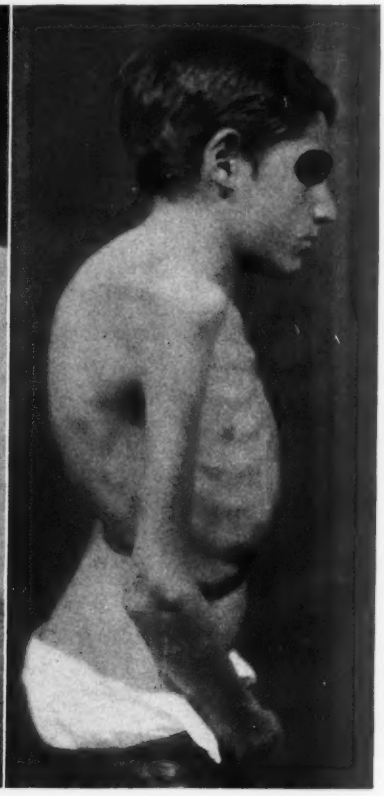


FIG. 10.

FIGS. 9 AND 10. Back and side views of patient in Group 3.

FIG. 11. Forward bending view of same patient as in Figures 9 and 10.

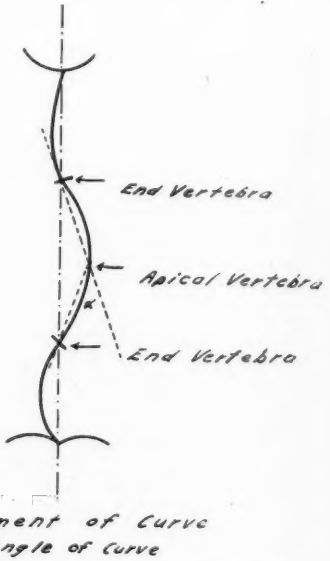


FIG. 12. Diagrammatic representation of method of curve measurement.

primary curve uncertain would be equally dangerous. To correct and fuse a compensatory curve would leave an imbalance in the

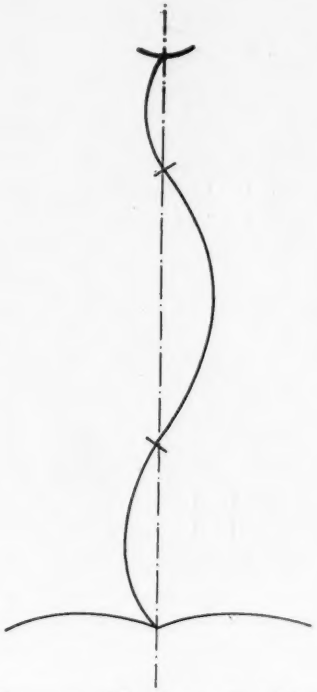


Fig. 13. Diagram of triple curve with middle curve primary.

primary curve, and the patient would have been far better off untreated.

The patients in the second group comprise probably the largest number of candidates for operative treatment. They consist for the most part of pre-adolescents whose deformity has been recently noted at school or at home and who have been sent for advice and treatment. Some have curves in the lumbar spine that can be completely corrected, but in most, the primary curve is higher, making the prognosis for complete correction somewhat less favorable. But if a patient with moderate deformity can be corrected to a point below which the deformity is apparent, and, at the same time, whatever displacement there is be completely corrected, a very good clinical result should be expected. These patients are told beforehand that although the roentgenogram will not show a geometrically straight spine, their backs should present little or no deformity even to the trained eye.

When we see a patient with a severe rigid

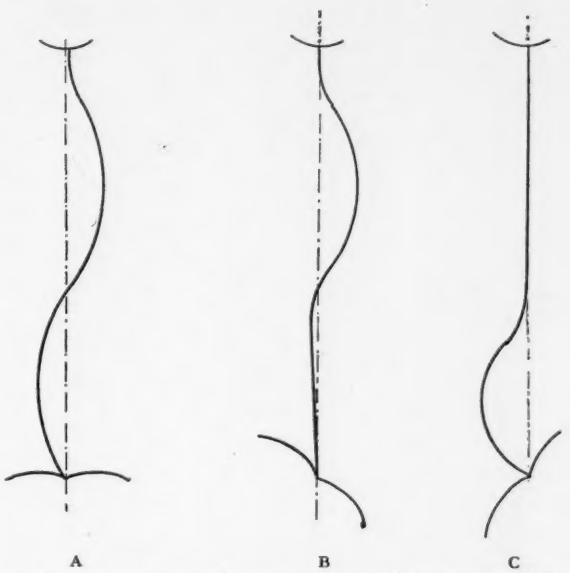


Fig. 14. A, type of curvature requiring study with pelvic tilt *x*-ray. B, behavior with thoracic curve primary. C, behavior with lumbar curve primary.

curvature, with its attendant structural changes in the vertebral column and thorax, as well as poor vital capacity and lowered general health, we should not give up in despair and say that since the spine cannot be appreciably straightened there is nothing to be done. These patients often have gone from clinic to clinic, and we sometimes see them suffering from mental abberation because of an inferiority complex due to the deformity. These individuals are told they will receive little or no correction of the curve itself but should obtain a reëstablishment of body balance that will greatly improve the general health and make the treatment a worthwhile process.

Often patients come in complaining of a painful area of the spine, and subsequent examination reveals the presence of scoliosis. Such patients often do not have deformities sufficiently severe to warrant a very long period of treatment. Exercises and massage are begun, and if these are not successful, fusion of the painful area is advised. Since the time in bed is not much

Fig. 15. A congenital curve which is partly due to a wedged fifth lumbar vertebra.

Fig. 16. Disappearance of the curve with elevation of the right side of the pelvis.

Fig. 17. Typical psoas curve taken in the sitting position.

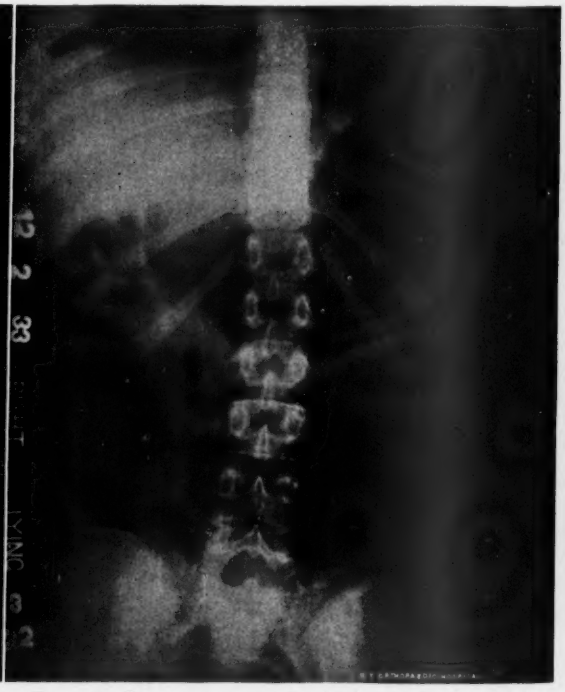


Fig. 18. Same curve with thigh on the left side actively contracted against resistance.

longer they are put through the regular correction treatment.

Measurement of a Curve. The accurate measurement of a curve is accomplished as follows; The apical vertebra and the ends or neutral vertebrae are determined. The complementary angle of lines drawn through the center of the bodies of these three vertebrae is designated as the angle of curve. This should be measured in the roentgenograms in both the erect and the recumbent positions. In all subsequent measurements the same vertebrae are used.

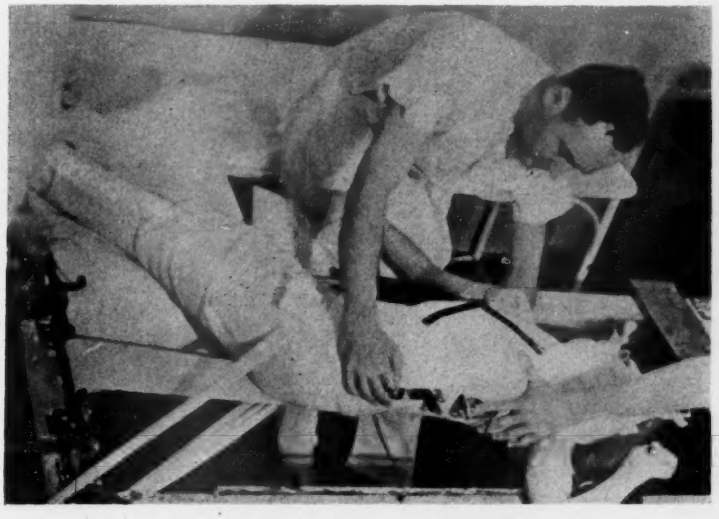


Fig. 19. Application of wedging jacket.

Fig. 20. The wedging jacket completed.

Fig. 21. Wedging begun.

With reasonable care the error should not be more than 3 to 5 degrees, slightly more when kyphosis is present. (Fig. 12.)

Determination of the Primary Curve. Before one can treat a case of lateral curvature successfully it is necessary to determine the primary curve and its ex-

tent. In cases with so-called triple curves this can usually be done by inspection. (Fig. 13.) These curvatures have either a

Fig. 22. Finished jacket after correction. Ready for fenestration.

return to the erect above and below the primary curve or a compensatory one is formed above or below the primary curve. If one considers the upper curve primary it is known that the spine does not form two compensatory curves below the primary and, conversely, if one considers the lowest curve primary there are not two compensatory curves above the primary. It is known, however, that a primary curve can form two compensatory curves, one above and the other below the deforming one. Hence in this case the middle curve is the primary curve.

In cases with double curves of approximately equal extent and severity the selection of the primary curve at times offers great difficulty. (Fig. 14.) In these cases it is necessary to take roentgenograms in the sitting position, with first the right and then the left side of the pelvis elevated. This is done to study the power of the muscles acting on the sides of the convexity of the two curves. If a patient with a right dorsal, left lumbar curve be seated with the left side of the pelvis elevated, the lumbar curve should correct for this pelvic tilt more than for the dorsal because of the

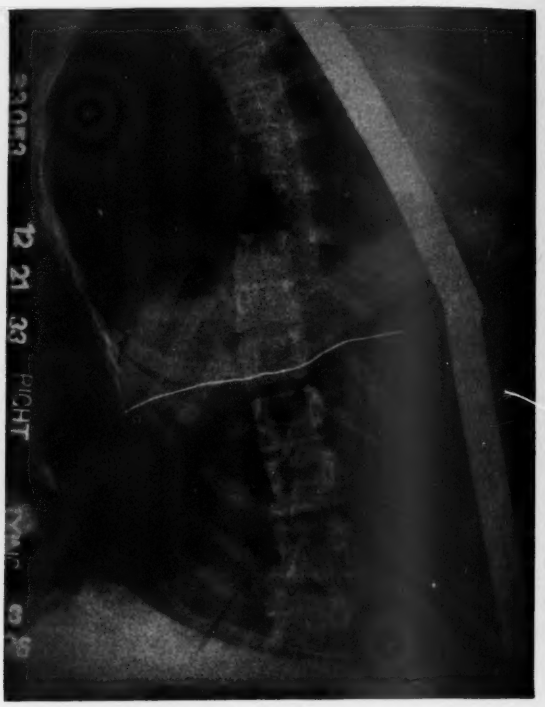


Fig. 23. Preoperative marker roentgenogram.

Dotted Line Represents Pelvic Tilt Film.
Solid Line Represents Spine in Maximum Correction

Fig. 24. Method of superimposition of pelvic tilt film and film of spine in maximum correction.

greater flexibility of the lumbar spine. If there is no ability in the lumbar spine to correct, the weight shifts to the right in the lumbar region and must therefore be shifted to the left in the dorsal in order to center the head over the pelvis. This causes

an increase in the lumbar curve and a decrease in the dorsal, showing a weakness of the muscles to the side of the convexity of the lumbar curve. Hence, the lumbar curve in this case is primary. Conversely, if the right side of the pelvis is elevated the power in the muscles acting on the right of the dorsal spine may be studied. The curve thus showing the imbalance is designated the primary curve. Rarely are primary curves seen. This does occur at times in scoliosis due to poliomyelitis.

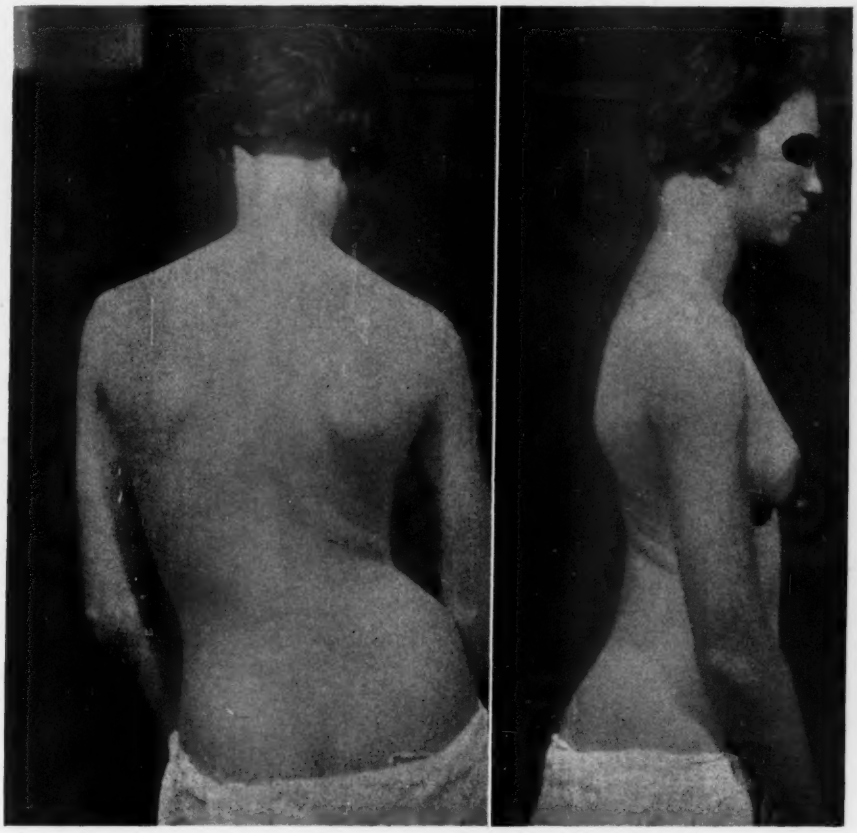


Fig. 25. Fig. 26.

Figs. 25 and 26. Case 1, before treatment.

At times we see a curve which may be thought to be due to a tilt of the fifth lumbar vertebra on the sacrum. To prove or disprove this it is necessary only to seat the patient with the superior surface of the fifth lumbar vertebra level. If the curve disappears the cause has been established. (Figs. 15 and 16.)

Curves due to psoas imbalance possess definite characteristics and should not be easily confused with other similar curves. (Figs. 17 and 18.) These psoas curves include only the five lumbar vertebrae and the entire curve is outside or lateral to the center line of the pelvis. These curvatures show very little rotation, and the spine above may return to the erect or show a slight compensatory curve. If the patient is placed on his back and the thigh on the concave side of the curve is actively flexed against resistance, thus creating a pull on the psoas muscle, the roentgenogram will demonstrate an increase in the curve, and, on the other hand, if the actively contracting psoas muscle on the convex side is likewise resisted the roentgenogram will show a straightening of the curve.

The Application and Care of the Wedging Jacket. The application of a comfortable, well-fitting and efficient jacket is one of the most important steps in the successful treatment of scoliosis.

A very simple apparatus is used, consisting essentially of a wooden frame with a

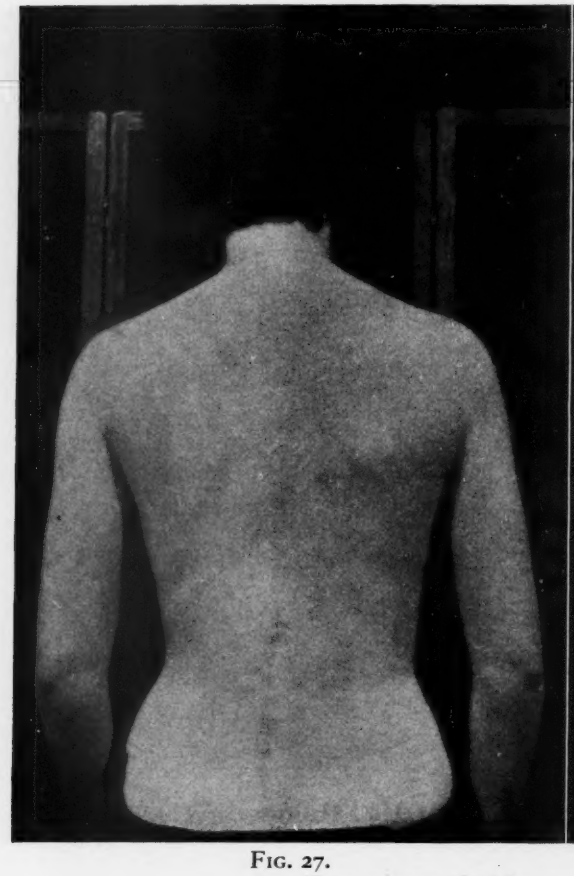


Fig. 27.

Fig. 28.

Figs. 27 and 28. After treatment.

Fig. 29.

Fig. 30.

Figs. 29 and 30. Case 1 in the sitting position before and after treatment.

removable steel crossbar in the middle. At each end is a windlass. One of these windlasses is used to hold taut a web strap

The hardware consists of two flat hinges and two turnbuckle lugs. The hinges may be made from ordinary strap iron, with the

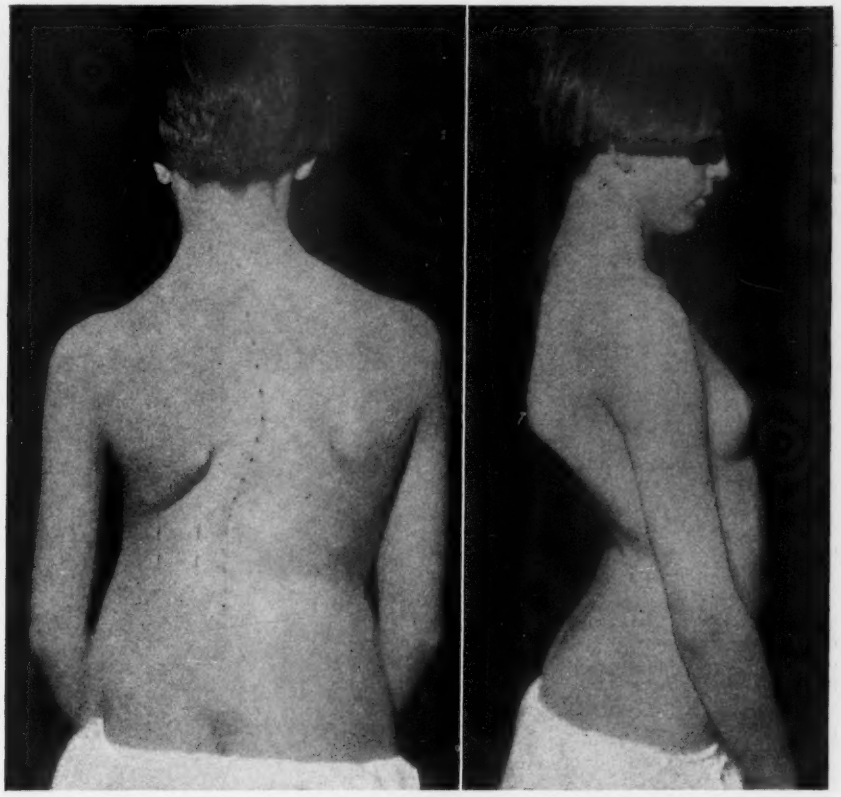


Fig. 31. Fig. 32.

Figs. 31 and 32. Case II before operation (April 20, 1927).

attached to the steel crossbar. This should be drawn up very tightly to minimize the sag when the patient is placed upon the frame. The windlass in the other end is used to hold the head in moderate traction during the application of the jacket.

The patient is invested with stockinette extending from above the head to below the knees. Holes are cut in the sides for the arms. The stockinette is tied in a crop over the head to prevent the hair from becoming soiled during the application of the plaster. A small hole cut over the nose allows for more comfortable breathing.

Pads of thick felt are then cut and placed to distribute pressure over bony prominences and points of pressure, such as occiput, chin, axillae, spine and about the pelvis. When thick felt is used a much smoother interior can be made if the edges are bevelled.

flat surfaces roughened and perforated by steel wire to give them a better grip in the plaster. The lugs as shown may also be made from strap iron.

The patient is then placed on the frame, lying in the web strap, with the crossbar at about the level of the seventh cervical vertebra. For orientation it has been found advisable at this point to place the roentgenogram directly on the patient's chest to lessen the possibility of bending the spine against the wrong curve and to visualize the spine better during the correction process. A sling made of two pieces of muslin bandage is placed over the head and attached to a spreader, which in turn is connected to a windlass. Sufficient traction is made on the head to take up the slack and at the same time maintain the head in a slightly extended position. This should be maintained steady by an assistant. The

trunk is then bent against the primary curve. With this a certain amount of correction is obtained. It is important that the pelvis be maintained level with the shoulders, and not rotated, during the bending process. The arm on the convex side of the curve under correction is brought to the side of the frame, while the other arm is extended over the head. At this point it is well to tilt the head about 10 degrees toward the concave side of the curve under correction, which will lessen the pull on the branchial plexus when final wedging is under way. To act as a counter-force a 6-inch muslin bandage is placed about the pelvis on the convex side of the surve to be corrected and this tied to the opposite side of the frame.

The felt pads are then held in place over each iliac crest, over the sternum, sacrum, spine, under the axillae, and about the head. The axillary pad on the convex side is split nearly through sagittally and the space filled with cotton to form a triangular wedge. This will prevent the cut edge of the jacket from causing a pressure line on the thorax after wedging is begun.

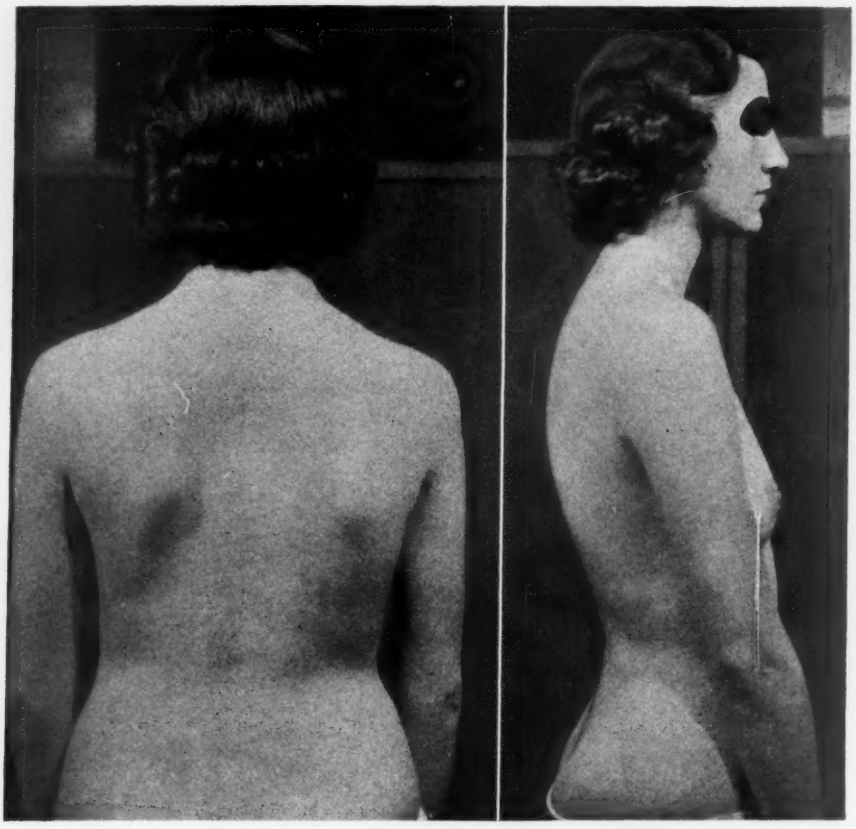


Fig. 33. Fig. 34.

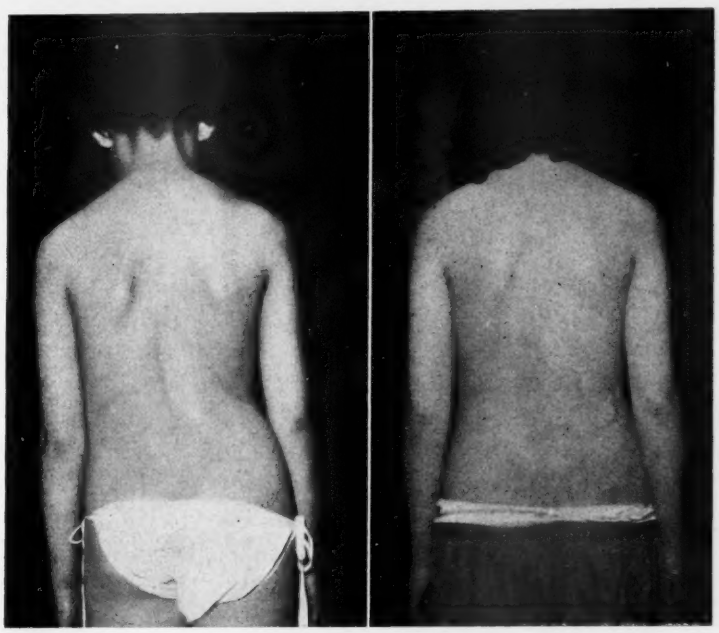
Figs 33 and 34. Case 11 (February 15, 1935), nearly eight years after operation.

After the placing of the felt pads, the application of the plaster is begun. One assistant starts at the pelvis while another works on the application of the head-piece. The pelvic portion should be wrapped snugly and moulded well about the crests, while the portion about the epigastrium and lower ribs should be laid on rather loosely. As the axilla is approached the bandages are drawn tighter. The head-piece must be laid on smoothly and moulded well about the jaw and occiput. After eight or nine layers have been applied the head-piece and body portions are joined together by plaster reinforcements or reverses. The hinges are then placed, the joints of the hinges at about the apex of the curve and

on a plane transverse to the trunk. Before incorporating the hinges in the plaster they should be bent to an angle of about the same degree as the primary curve so that as correction progresses they will straighten out. The turnbuckle lugs are incorporated into the jacket at about 3 or 4 inches below the joints of the hinges and on the concave side of the curve under correction. The hardware should be well incorporated in the plaster and nearly smoothed off. Before the plaster sets some sort of a mark is

Fig. 35. Preoperative roentgenogram of Case II.

Fig. 36. Roentgenogram of Case II taken nearly eight years after operation.



A B

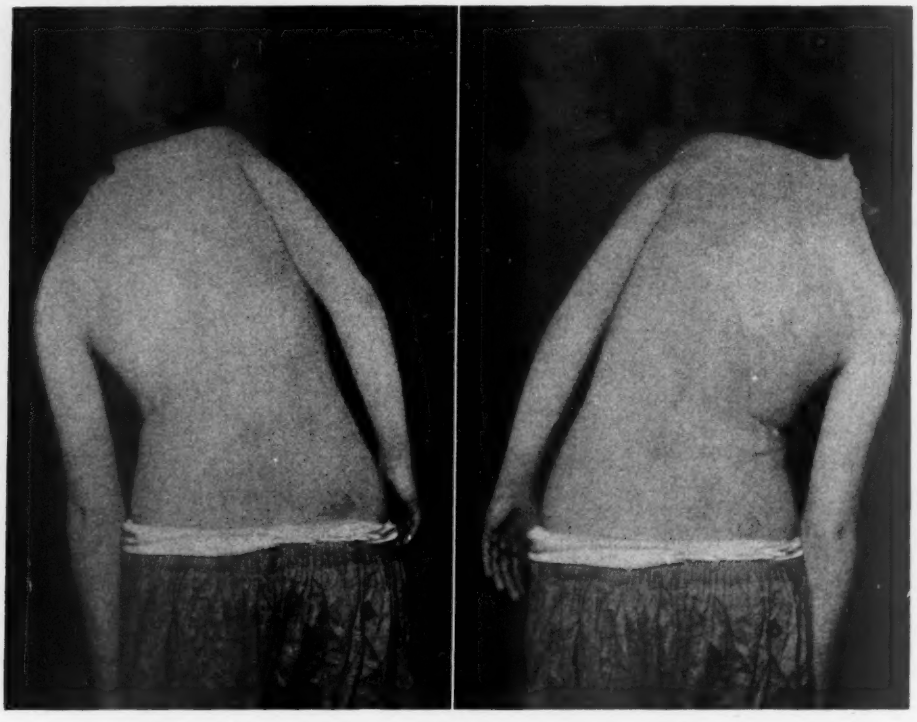
Fig. 37. Case III. A, on January 24, 1928. B, on January 5, 1935.

placed on the surface of the jacket to identify the joints of the hinges. (Fig. 19.)

With the patient still in this position on the frame, the thigh on the convex side of the curve is flexed about 10 degrees and abducted about 20 degrees. The remaining stockinette is cut and smoothly placed about the thigh. The lower portion of the jacket over the same iliac crest is cut upwards for about 4 inches into three or four strips about 1½ inches wide. This will relieve any pinching that may have occurred during the application of the pelvic portion. These strips are separated into several layers and folded in, one to each turn of the plaster as the spica is being applied. If greater strength is required a basswood splint may be incorporated.

The patient is then carefully removed from the frame and the edges of the jacket trimmed and bevelled. The head-piece should be trimmed wide enough to give room for the ears and the mastoid processes. (Fig. 20.)

After the patient has been returned to bed, a free circulation of air should be maintained to dry the jacket thoroughly. A gastric upset may at times follow, but this is temporary and subsides quickly as the patient becomes accommodated to his new position.



A B

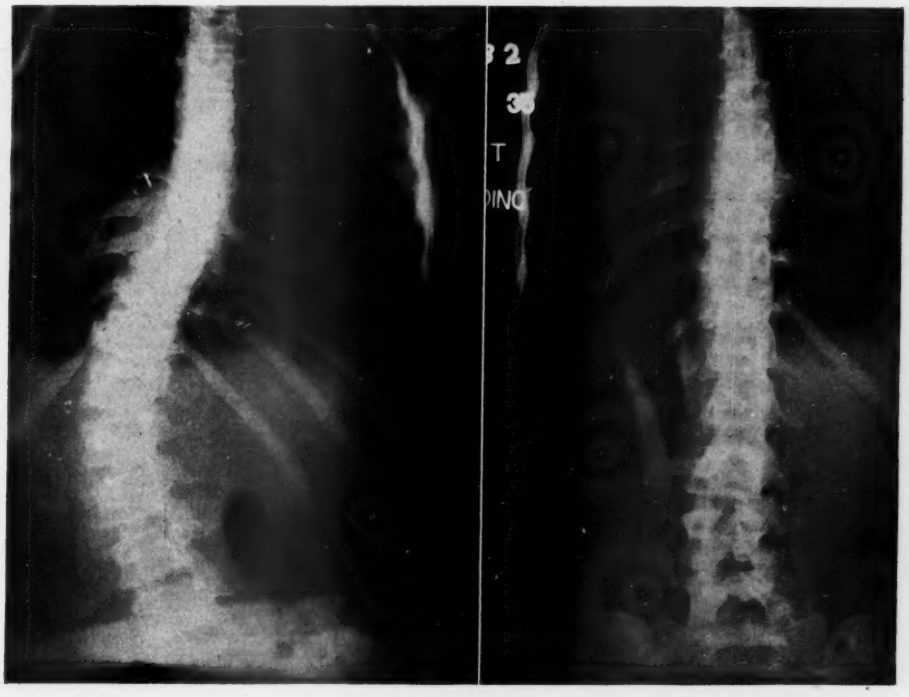
FIG. 38. Case III, showing lateral flexibility of spine.

The care of the jacket at this time is especially important, and nurses and interns should be instructed to inspect it frequently and keep a sharp lookout for pressure areas, which must be promptly relieved. Any area of burning or numbness complained of should be promptly investigated and if this persists a small fenestration must be made to relieve the pressure.

Wedging. After seven to ten days the jacket has become sufficiently dry and strong for wedging. A segment is removed on the convex side about 3 inches wide at the center, tapering off to the joints of the hinges. On the concave side a curved cut is made connecting the joints of the hinges and the interval between the turnbuckle lugs. The upper section is then swung on the lower and a small turnbuckle is inserted. This turnbuckle is replaced by a larger one when the limit of the smaller has

been reached, and extended as much as the patient can stand comfortably each day. The wedging is best done in the morning so tension can be relaxed if necessary in the evening. With reasonable care a curve can be corrected and the patient kept fairly comfortable during the entire process. The maximum amount of correction obtainable can be learned from roentgenograms taken at intervals. One should direct his attention during the wedging to the arm on the concave side for evidence of pull on the cervical nerve trunks, manifested by anesthesia and weakness. If this does occur the neckpiece may be cut through and the head tilted still more to give relaxation. (Fig. 21.)

After the optimum correction and compensation have been obtained, as determined by roentgenographic examination, the upper and lower sections are well padded over the exposed portions of the thorax and plaster bandages are then used to join these into a solid unit. Plaster-covered basswood splints are then placed about the back to give further strength, securely fastened at their ends. (Fig. 22.) The next day the posterior portion of the jacket may be cut out to expose the operative area. The stockinette is bound to the edges of the fenestration with adjesive and the skin thoroughly cleansed. A silver nitrate mark is placed on the skin over a spinous process in the primary curve to serve as a guide at operation. This is accurately identified by a roentgenogram taken with a small piece of wire over the the silver nitrate marker.



A B

Fig. 39. Case III. A, roentgenogram, 1928. B, roentgenogram, 1935.

Before the iodine is placed on the skin at operation, if the marker is not very clear it is well to reinforce it with gentian violet stain. The operative field is now ready for preoperative preparation. (Fig. 23.)

Selection of Area for Fusion. After the primary curve has been corrected in the wedging jacket, the selection of the proper area for operative fusion of the spine is a matter requiring the exercise of much judgment and it is here that errors are most likely to be made. At times, what should be a good result can be completely nullified by the addition or subtraction of even one vertebra at the ends of the fusion area,

especially if the patient be a growing individual.

In the ideal case, when the entire primary curve has been completely corrected, all the vertebrae should have their transverse axes parallel, and intersect the vertical axis of the trunk at right angles. This being the area of imbalance, it is now only necessary to maintain this position to obtain complete restoration of the alignment of the spine. After a fusion in such a case the spine outside the fusion area should balance the trunk with no external assistance.

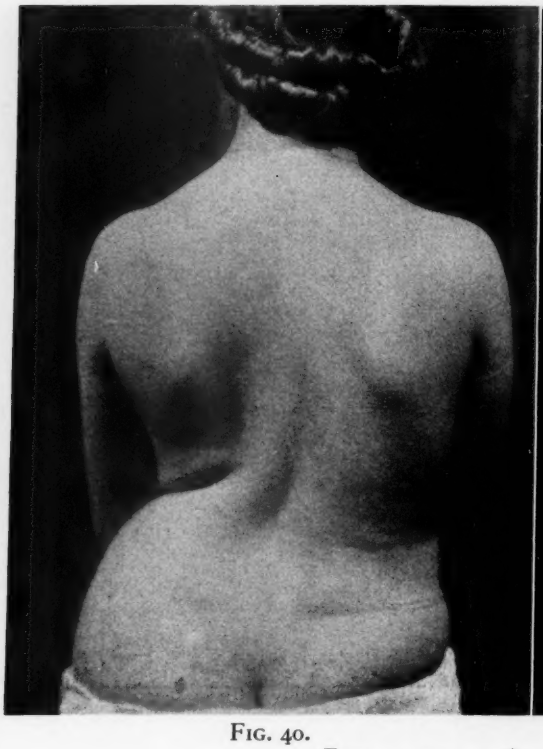


Fig. 40. Fig. 41.

Figs. 40 and 41. Case IV before correction.

In other cases, where complete correction has not been obtained and the transverse axis of the end vertebrae of the primary curve are not parallel to each other and at a right angle to a line joining their centers the above does not hold. In such cases to fuse only the primary curve would leave too much tilt in the vertebra at the ends of the fusion area for the compensatory curve or curves to take care of. Now if we examine the vertebrae beyond the ends of the primary curve we can, in most cases, locate two vertebrae, one at each end, whose surfaces are parallel to each other and at right angles to a line joining their centers. Therefore, in this case the fusion should be done to include not only the primary curve, but, in addition, sufficient vertebrae beyond the ends of the primary curve to give a level platform inferiorly, give symmetry to the trunk and enable the patient to stand erect without a list or a displacement. When the superior surface of the vertebra at the upper end of the fusion area is level the patient will hold his shoulders level with no effort.

In cases where there is severe structural wedging in the vertebral bodies of the compensatory curve or curves one must determine their flexibility before the application of the plaster jacket. This is done by means of roentgenograms taken with the pelvis elevated on the same side as the convexity of the compensatory curve, or it may be done with the spine bent laterally against the convexity of the compensatory curve. This film will show the amount of straightening the compensatory curve is

able to do when the primary curve has been corrected as much as possible. If we include in the fusion area the vertebrae at or vertically with the first thoracic vertebra over the first sacral vertebra and the film of the spine in maximum correction also

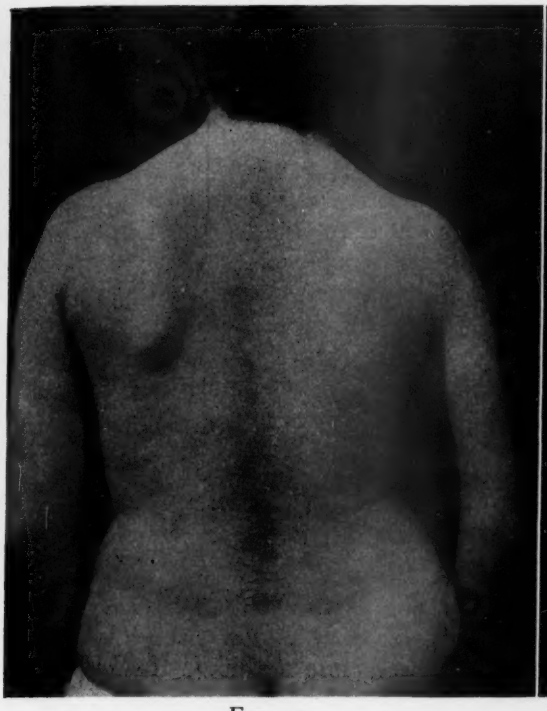


Fig. 42.

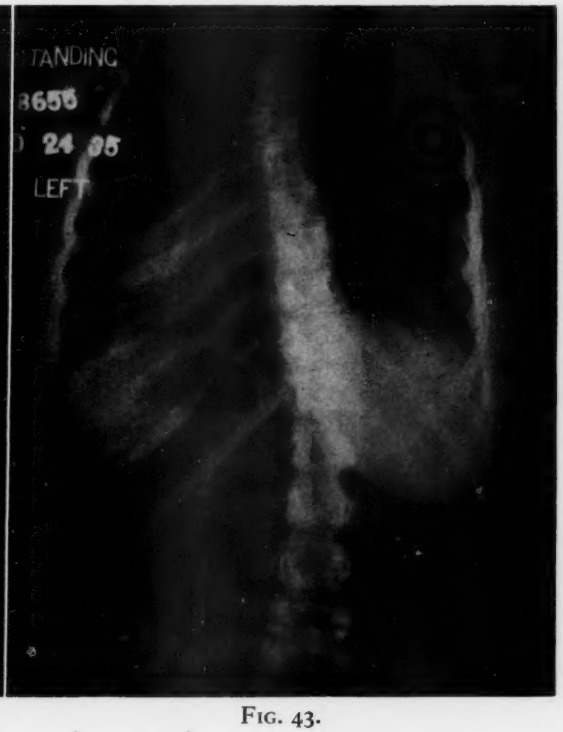


Fig. 43.

Figs. 42 and 43. Case IV after correction.

beyond the ends of the primary curve whose surfaces are parallel to each other and at right angles to a line joining their centers, the compensating portions of the spine should straighten or return to the erect in order to balance the trunk. When there is structural wedging or other hindrance to the compensatory portion or portions of the spine to straighten, fusion to a level vertebra inferiorly would give overcompensation of the curve. To arrive at the correct area for fusion in these cases the following method is used:

Place the film of the patient in the jacket, when in the position of maximum correction, over the film taken with the pelvis elevated on the side of the convexity of the compensatory curve or the film taken bent against the convexity of the compensatory curve. These superimposed films should give a fair idea of how the spine will look when the treatment has been completed. The pelvic tilt film should be placed vertical. These films are superimposed so that the lower end vertebra of the primary curve in each film coincides. The first vertebra at or beyond the end of the primary curve whose inferior surface coincides with the same surface of a corresponding vertebra in the other film should be taken as the lower limit of the fusion area. This will leave sufficient tilt in the transverse axis of the vertebra at the lower end of the fusion area to allow the compensating portion of the spine to balance the trunk. (Fig. 24.)

The Fusion Operating and After-Care. The spine is fused by the usual Hibbs method. Particular attention is paid to the apex of the curve, where the fusion is reinforced with chips from the spinous processes from the ends of the fusion area. When six or more vertebrae are to be fused the operation is carried out in two or more stages, about three weeks apart. The fenestration in the jacket is closed with

plaster after the last operation and the patient is kept in bed for twelve more weeks. After operation the patient may relax and suspend himself in the neck-piece. Careful observation will disclose points of pressure about the head and arms. Trimming to relieve this will not materially affect the correction.

When the jacket is removed roentgenograms are taken to determine the extent and maturity of the fusion, as well as the angle of curve. A light body jacket is then applied, with the head held lateral to the crest of the curve on the convex side in order to minimize the gravity as a deforming force. The patient is allowed up in this jacket. The jackets are changed at intervals of twelve weeks, the head being brought closer to the midline with each plaster change. In most cases nine months is sufficient time for the fusion to become mature enough to maintain the correction without external assistance.

CONCLUSIONS

The treatment of scoliosis by this method is long and difficult, but has given better results than the previous ones used at this clinic. This and other studies made on this subject have enabled us to state the following with reasonable accuracy;

1. If the fusion is solid and adequate in extent there will be no increase in curvature.

2. A loss in sitting height and an increase in curvature suggest a pseudarthrosis. Pain and localized tenderness also suggest a pseudarthrosis.

3. If there is no increase in curvature in the fused area but a definite increase in the curvature at the end of the fusion, with a loss of compensation, the fusion area is too short.

ILLUSTRATIVE CASES

Case I. M. K. was admitted to the clinic August 18, 1934, at the age of 15. (Figs. 25–30.) Wedging jacket was applied October 14, and the patient was ready for operation on November 1. Spine fusion was done in two stages from the ninth thoracic to the fourth lumbar vertebra. All support was removed August 8, 1935. Balance and symmetry have been restored and the spine is now geometrically straight.

Case II. E. P. was admitted to the clinic April 18, 1927. The curve had been noticed for four months. The right thoracic curve was primary and was corrected in a wedging jacket from 27 to 23 degrees. Fusion was done from the fourth thoracic to the second lumbar vertebra and has maintained the balance and secured improvement in posture. Now the clinical appearance of the trunk is excellent.

Case III. C. R. was admitted to the clinic at the age of 11, July 9, 1928. The lumbar curve was primary and was corrected in a wedging jacket from 37 degrees to zero. The spine was fused from the tenth thoracic to the fourth lumbar vertebra. Fusion should have included the fourth lumbar, but due to the over-correctness inferiorly the fusion was shortened purposely to give more lumbosacral flexibility and at the same time not have the spine become solid when the lower end vertebra is in an over-corrected position.

Note the excellent maintenance of correction and the good lateral flexibility of the spine below the fusion area.

Case IV. J. R., age 15 years had a curve first noticed in 1930. She was admitted to the clinic in 1934. Correction in a wedging jacket was accomplished from 44 degrees to 11 degrees. The spine was fused in two stages from the seventh thoracic to the third lumbar vertebra. All support was removed thirty-six weeks after operation. Note the remarkable correction of the curve and the improved appearance of the trunk even though complete correction has not been obtained.

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TUMORS OF THE MANDIBLE

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MANDIBULAR tumors form an increasing group of neoplasms, many of which are histologically and radiographically quite unlike any found elsewhere in the body. Clinically, it is well nigh impossible to separate the various types. However, the roentgenogram is responsible for the most accurate means of recognizing and differentiating these growths. Neoplasms of the jaw are most frequently classified according to their histologic elements into epithelial, osseous and dental tumors. In this way their division is based on the structural origin of the neoplasm. From the standpoint of behavior and treatment, such a classification proves of little clinical importance.

A classification that is based on the clinical course and which suggests the proper management of the growth is certainly of more practical value. With their separation into three general groups, tumors of the mandible may be benign, malignant, or benign with the tendency to assume malignant characteristics. These clinical divisions are not hard and fast, as certain benign growths may show a tendency to recur locally when not completely eradicated. This possibility may also affect benign growth elsewhere in the body.

Mandibular tumors may accordingly be divided as follows:

I. Benign Tumors
   1. Dental root cyst or radicular cyst
   2. Follicular or dentigerous cyst
   3. Odontoma
   4. Giant cell tumor and epulis
   5. Ossifying fibroma
   6. Osteoma or exostosis
   7. Osteochondroma
   8. Hemangioma

II. Benign Tumors Which Sometimes Take on Malignant Characteristics
   1. Adamantinoma or adamantine epithelioma
   2. Radiation osteitis—from radium or *x*-rays—resulting in sarcoma

III. Malignant Tumors
   A. Primary:
      1. Osteogenic sarcoma
      2. Endothelial sarcoma or Ewing's sarcoma
      3. Chondrosarcoma
   B. Secondary:
      1. Carcinoma
         (*a*) Epidermoid carcinoma—by direct extension from adjacent structures as the mouth, skin and glands
         (*b*) Adenocarcinoma—by metastasis from a primary lesion elsewhere in the body.
         (*c*) Melanotic carcinoma
      2. Sarcoma
         (*a*) Osteogenic sarcoma
         (*b*) Melanotic sarcoma
      3. Hypernephroma

BENIGN TUMORS

These mandibular tumors are slow-growing, hard, practically painless growths which, because of the facial deformity they produce, ultimately bring the patient to the physician. They occur at any age, but more commonly in children and young adults. Treatment is surgical; complete excision with cauterization is necessary to prevent local recurrence, for which they show a tendency. They never metastasize. Irradiation has little, if any, effect.

Radicular or Dental Root Cysts. Dental root or radicular cysts are the most common of the dental tumors. The cyst forms in the osseous structure of the jaw about the root of a devitalized tooth following chronic inflammatory changes. Although

there are no symptoms in the early stages, the larger cysts result in a noticeable swelling and expansion of the mandible.

Fig. 1. Radicular cyst of the mandible.

The roentgenograms (Fig. 1) show a rarefied or a cystic area in the body of the mandible with clear-cut borders extending about the root of the devitalized tooth, provided the dental structure has not been removed at some time previous to the examination. There is no evidence of bone proliferation around the cystic space. These growths are to be distinguished from an ordinary apical granuloma which is usually smaller than a radicular cyst and has indistinct hazy margins, and from a follicular or dentigerous cyst. The latter is diagnosed by the presence of a nonerupted tooth with the cystic formation about its crown rather than about the apex of an erupted tooth, as occurs in radicular cysts.

Dental root cysts are occasionally multiple, involving the roots of several neighboring teeth. They then assume a multilocular appearance and must be distinguished from other multilocular cyst formations.

Follicular or Dentigerous Cysts. This type is rare and occurs usually in children under 15 years of age. The most common site is in the region of the third molar tooth. The jaw shows slow enlargement at the site of the tumor and the tooth fails to erupt. The roentgenogram reveals a unilocular cystic area with sharply defined walls about the crown of an unerupted tooth. Bone production about the cystic space is absent.

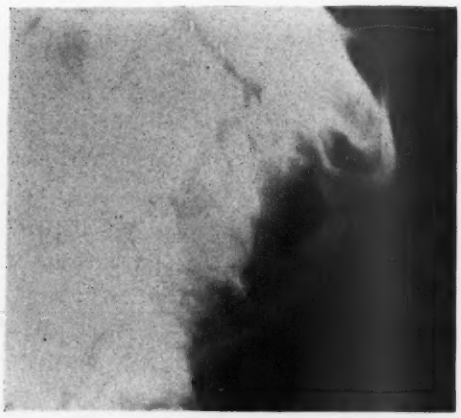

Fig. 2. Composite follicular cysts. Cysts are multilocular and one contains an unerupted tooth (arrow).

Rarely, the cyst assumes a multilocular appearance due to the superposition of several cysts involving the crowns of unerupted teeth, similar to the formation of multilocular radicular cysts. These are then spoken of as *compound* or *composite follicular cysts.* (Fig. 2.)

Pathologically, these cysts arise from the epithelium of the enamel organ during the development of the teeth.

Odontomas. These are mixed tumors occurring more frequently than follicular cysts. They are usually of bony hardness, appearing in the lower jaw of children or young adults at the site of an unerupted tooth, or from accessory tooth germs that adjoin the unerupted follicle (*composite odontoma*).

Roentgenologically, odontomas are characterized by irregular, densely classified, compact masses within a cystic space which is well delimited by normal bone structure, showing no proliferative changes. (Fig. 3.)

Giant Cell Tumor and Epulides. Central giant cell tumors of the mandible usually grow rapidly and produce early facial deformity. The Roentgen examination (Fig. 4) shows a polycystic area of bone absorption with clear-cut margins. The jaw is expanded and the cortex thinned out. At times, especially in larger lesions,

radiation therapy is necessary to augment the effects of curettage and cauterization, either to prevent spread or to control a recurrence. The danger of radiation osteitis must always be borne in mind when irradiating with heavy doses in and about the jaw.

Fig. 3. Odontoma of the mandible.

There are other neoplasms which particularly affect the gums and the alveolar ridge, and are designated as epulides. They are commonly found during pregnancy when they tend to grow rapidly. The more frequent pregnancy tumors (also designated as pregnancy gingivitis or epulis gravidarum) are the angiomas and granulomas; osteomas, fibromas and alveolar giant cell epulides are less common. These gingival neoplasms exert pressure on the teeth, the displacement of which is clearly observed on the films.

Ossifying Fibromas. These benign tumors are made up of fibrous tissue, bone and sometimes cartilage. Roentgenologically, they show a coarse mottling within a regular delimiting area which appears less dense than normal bone. They have no cystic space or sharp trabeculations to confuse them with the polycystic tumors, and their slow growth and distinct contour help to exclude a malignant condition.

Osteomas or Exostoses. Osteomas are the more differentiated and less cellular form of ossifying fibroma. The roentgenogram shows a small dome-like swelling of increased density. The prognosis is good, and, in most cases, surgery is not imperative.

Fig. 4. Central giant cell tumor.

The more spongy growths may be chiseled from their base without the likelihood of recurrence.

Fig. 5. Hemangioma, capillary type. Note the many small cystic spaces like pouched-out areas in the bone, produced by the dilated capillaries or smaller blood spaces.

Osteochondromas. An osteoma containing a layer of cartilage is recognized as an osteochondroma. The dense mass in the roentgenogram shows a spotted or irregular appearance due to the radiotranslucency of cartilage. Treatment is the same as for osteomas or exostoses.

Hemangiomas. This type is a very rare, benign growth which may not only be present in the bone of the mandible itself, but may also be found to invade the gingival or buccal structures at the same time. As elsewhere in the body, it assumes two different forms depending on its histology. The cavernous type shows large spaces of diminished bone density, often with fine radiating spicules or long thin strands of bone in the rarefied area. In the second or the capillary form (also called the scirrhous type), the roentgenograms (Fig. 5) show many small cystic spaces which are produced by the dilated capillaries or smaller blood spaces scattered through the bone. In the more developed stage, it suggests the Roentgen appearance of osteitis fibrosa cystica.

Fig. 6. Adamantinoma of the mandible.

Bone hemangiomas respond to irradiation, ossifying slowly following treatment.

BENIGN GROWTHS WHICH SOMETIMES TAKE ON MALIGNANT CHARACTERISTICS

Adamantinoma. Adamantine epitheliomas are unusual and interesting epithelial growths, potentially malignant, which take their origin from the enamel and dentine organs. Less common than radicular cysts, they occur more frequently than follicular cysts. Adamantinomas are slow-growing, practically painless tumors which most often affect young adults and negroes, and gradually produce facial asymmetry which ultimately brings the patient to the physician.

The roentgenogram (Fig. 6) usually shows bone destruction with the formation of a number of conglomerate cystic spaces and a thinned out cortex. A faint irregular mottling can be made out in the cystic areas. Occasionally, there is a central cystic tumor of sharp outline, not unlike that produced by the giant cell tumor or the radicular or follicular cyst. In many cases, the teeth are displaced by the expan-

sion of the growth and sometimes a tooth follicle is present in the cyst. An absolute diagnosis cannot always be made as, not infrequently, any of the central tumors may show a similar picture. The adamantinomas produce no periosteal reaction, such as is seen in sarcoma. They have not the worn, eroded edges of the cancerous growths or the bone proliferation observed in osteomyelitis.

Adamantinomas may be cystic or solid and are frequently lobulated. Even when solid, they show a cystic or rarefied appearance on the Roentgen films. Treatment consists of curettage and cauterization. They have a strong tendency to recur when they may attain considerable size. Like the benign neoplasms, these tumors are radioresistant. Even with one or more recurrences, the prognosis is good in most cases. Metastasis is rare, but death has occurred from direct extension to the skull and brain.

Aberrant adamantinomas have been recorded in the ovary (where they are teratomas), tibia and the hypophyseal duct.

Fig. 7. Osteogenic sarcoma. Note the spicule formation lying at right angles to the maxilla.

Radiation Osteitis. Although this condition is not neoplastic, excessive radiation to the jaw may result in bone necrosis, which, in certain rare instances, may undergo sarcomatous changes. Such malignant development has been particularly observed in industrial workers with radium poisoning. Whether a similar complication is possible in radiation osteitis following intensive roentgen therapy remains a mooted question.

PRIMARY MALIGNANT TUMORS

Osteogenic Sarcoma. Primary involvement of the mandible with sarcoma is rare.

Almost any pathologic type has been known to occur in the maxilla. They usually grow rapidly, producing early facial asymmetry, and are fatal regardless of treatment. Extensive skeletal metastases may occur early in the course of the disease. In the sclerosing type, growth is exceedingly rapid with the average duration of symptoms about two months. The roentgenograms (Fig. 7) show irregular dense foci of new bone production alternating with areas of bone destruction. The margin of the neoplasm shows a periosteal reaction with occasional spicule formation extending at right angles to the maxilla.

FIG. 8. Secondary involvement of the lower maxilla from a hypernephroma.

Endothelial Sarcoma. Ewing's sarcoma usually occurs in children and young adults. The symptoms of pain and swelling of the jaw are brief, frequently of one month's duration, an unusually rapid course for lesions of the jaw. The roentgenograms show irregular areas of bone destruction with or without expansion of the cortex or periosteal new bone. The findings are not characteristic. Although the lesion is radiosensitive, cures are indeed rare.

Chondrosarcoma. This tumor is an osteogenic sarcoma containing cartilage. Although growth is less rapid than in the other types already mentioned with a year or more elapsing before treatment is sought by the patient, the outcome is fatal regardless of the form of therapy. Repeated local recurrence is the rule before distant metastasis takes place. Roentgenologically, an area of osteoporosis is present, extending rapidly either to the alveolar or to the lower margin of the mandible. Erosion takes place without expansion of the bone, distinguishing these growths from benign tumors.

METASTATIC TUMORS

The jaw may be invaded with malignant growth by the blood stream, lymphatic channels or by direct extension. Intra-oral epidermoid carcinoma, especially from the gums, floor of the mouth, tongue, and lips, not infrequently extends to the bone which becomes irregularly eroded by the cancerous invasion. Secondary involvement of the mandible from a primary sarcoma or carcinoma may rarely occur. The more likely primary malignant seats are the prostate, thyroid, ovary, testicle, kidney (Fig. 8), melanotic lesion usually of the skin, breast and rarely the stomach. Mandibular involvement has been observed in multiple myeloma and lymphoblastoma.

DIFFERENTIAL DIAGNOSIS

Mandibular tumors may not infrequently be confused with the following non-tumorous conditions:

1. Technical factors:
   (*a*) Superimposition of the hyoid bone over the mandible.
   (*b*) Calculus in the salivary gland.
   (*c*) Air in the pharynx.
2. Osteitis fibrosa cystica or hyperparathyroidism.
3. Osteitis deformans or Paget's disease (leontiasis ossium).
4. Syphilis.
5. Tuberculosis, actinomycosis, blastomycosis.
6. Radiation osteitis.
7. Osteomyelitis.

Faulty technique in regard to positioning of the patient and the tube may superimpose the hyoid bone and air-containing pharynx over the mandible, with the result that a cystic lesion of the jaw is simulated. A salivary calculus overlapping the maxilla may suggest the diagnosis of an odontoma or an exostosis. Changing the position of the patient or tube will tend to separate the shadow cast by the stone from the margins of the jaw.

Rarely, Paget's disease may be manifested in the jaw (leontiasis ossium of the face) for many years before the rest of the skeleton becomes involved. The cystic lesions observed in hyperparathyroidism may also attack the mandible, but this involvement is usually late in the course of the disease which is considerably advanced by this time and shows widespread evidence of its presence.

Syphilis in the form of a gumma may rarely involve the jaw. Tuberculosis, actinomycosis and blastomycosis have been observed to invade the mandible from intra-oral lesions which are recognized by examination and biopsy.

Radiation osteitis, the result of exposing the jaw to intensive radium and *x*-ray bombardment, reveals itself as a necrotic lesion. It is more apt to occur in the presence of infection, particularly of the teeth, and results in changes not unlike those observed in osteomyelitis. As a prophylactic measure, oral sepsis should be eradicated as thoroughly as possible and diseased teeth removed before executing any plan of intensive radiation therapy in the region of the jaws.

Osteomyelitis is of rather frequent occurrence and, roentgenologically, may resemble a new growth. Usually the history of a systemic reaction with an elevation of the temperature, chills and malaise helps to separate the two conditions. In infections of the mandible, a predominant symptom is pain which is seldomly very pronounced in neoplasm.

SUMMARY

A classification of mandibular tumors is suggested, based upon clinical behavior. The various types of neoplasms invading the lower jaw are described, and an attempt is made to differentiate them from one another as well as from non-neoplastic conditions.

SOME COMMON ERRORS IN DIAGNOSIS AND TREATMENT OF CHEST PROBLEMS*

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PHYSICIANS and surgeons who have not followed intimately during the past few years developments in the field of chest surgery are frequently lacking in concepts of diagnostic and therapeutic possibilities in this field. This statement is not necessarily a condemnation of any part of the profession, but simply a reference to an unavoidable stage through which medicine must advance in its forward progress.

Those familiar with more modern methods of treatment and diagnosis in the chest surgery field are frequently surprised that prominent internists will often treat a patient with, for example, a basically unimproving lung abscess by so-called conservative measures for a long period, when every modern indication calls for adequate surgical bronchoscopic or surgical drainage relatively early in the disease; or that a frank lung abscess or an empyema may be treated for weeks by well known general practitioners with a diagnosis of "unresolved pneumonia."

Nevertheless, instances such as these, as every chest surgeon knows, are occurring daily. And the deplorable part of this situation is that actually very little, relatively speaking, is being done to narrow the wide breach which exists between the type of diagnosis and treatment known to men especially interested in chest pathology and that familiar to the average surgeon or physician not especially practiced in this field.

There are a number of reasons for this wide discrepancy in our efficiency, the discussion of which would not add greatly to the value of this presentation. One of these reasons, however, is that the men especially interested in following the rising star of thoracic surgery have become more or less organized in special groups. They tend to discuss their problems in special meetings and in journals devoted exclusively to the study of thoracic disorders. This course of action is a desirable one for the most part, enabling those who are pioneering the field to progress more rapidly than if chest problems were developed in less concentrated fashion. However, there is also a definite need for wider publication in journals devoted to broader phases of medicine and surgery in order that applicable fundamentals in chest problems may reach a larger portion of the profession.

A few illustrative case reports with relevant discussion are therefore presented here. There is little in the case reports or the discussion which will be of specific interest to a specialist in thoracic surgery. That there is material here, however, which should be of interest to a great many physicians and surgeons is indicated by the several errors in judgment or procedure on the part of physicians or surgeons who ordinarily are quite efficient in the handling of other human ailments. No further reason need be offered for publication of a paper which is intended, not as a contribution of original advances in the field, but as an appeal to a wider appreciation of basic factors already accepted in special chest surgery circles.

These cases represent a part of the routine work in the Chest Surgery Division of the University of Arkansas School of Medicine.

CASE 1. E. G., a white male of 46 years, was admitted November 11, 1936, with a history of

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"influenza" ten months previously. Following this he continued to have a moderately productive, non-odoriferous cough and some aching pains in the right chest. The chest had been *x*-rayed shortly after the attack of "flu" and the patient had been told the picture "showed nothing." Afterward he went to a well-known internist who told him that he had a lung abscess and *treated him with "conservative measures" for a period of several months*, i.e., until three weeks prior to his admittance to the hospital.

Upon his admission, 900 c.c. of thin, green, putrid fluid was obtained by aspiration through the ninth space in the posterior axillary line. The patient, with a diagnosis of empyema, was then transferred to the chest surgery service with the internist's recommendation that Mozingo drainage with or without rib resection be instituted. Here, 800 to 900 c.c. of pus, similar to the first, was obtained by aspiration from one area of the chest and about 400 c.c. was aspirated from another area.

The pus was smeared and cultured and *organisms belonging to streptothrix group obtained in pure culture*. After consultation with Dr. Wohlin of the bacteriology department a working diagnosis of pulmonary actinomycosis was made.

X-ray (Fig. 1) examination on November 13 showed three different fluid levels in the chest. A thoracotomy was done on November 25. Three separate and distinct cavities in the pleural cavity were opened. Thick old adhesions firmly and completely separated the right pleural cavity into two large anterior and posterior cavities by running from the dome on the right chest down the lateral wall, ending apparently by branching into an inverted Y with the two prongs forming another small but complete cavity in the lower lateral aspect of the right chest. About 700 c.c. of yellowish purulent odoriferous fluid was obtained as the septa were broken down. No sinuses were seen in the pulmonary tissue proper. Drainage tubes were installed in the pleural cavity.

Though little assistance was expected from massive doses of iodides and *x*-ray therapy, it was felt these latter agencies were possibly indicated as a temporary measure because of the streptothrix micro-organisms. Iodides were administered, but it was necessary to discontinue them in a few days because of intolerance. For the next thirty days, the patient with good drainage of the chest, showed definite clinical improvement, gained weight, and felt better. On December 27 it was noted that an obstructive type of inspiration, moderately labored had been noticeable for three or four days and coughing had increased. Definite sonorous and sibilant râles were heard over the right chest. The cough was essentially non-productive and the general condition was fair.

Fig. 1. Case 1. All separate pockets of pus which were present are not discernible here, but this oblique view clearly illustrates two separate fluid levels. The operative notes given in the first and fourth cases reported clearly signify the necessity of realizing that several separate pus pockets are sometimes present in empyema; and that all these pockets may not be drained by the simple insertion of a single tube into the pleural cavity.

Drainage from the chest was decreasing, but apparently the lung was not expanding. On December 30 the cough became more severe. Morphia was necessary for its control at times. The breathing was labored and of a type which indicated partial obstruction of the left main bronchus. It was believed that granulation tissue was filling in from the right bronchus or that possibly a tumor was present. The patient was obviously losing ground and other procedures were indicated, the first of them a bronchoscopic examination. It was in our minds at this time to confirm or rule out new growths or remove any granulation tissue which might be associated with the suspected actinomycosis of the lung, and then consider pneumonectomy.

Bronchoscopic examination done on January 5, 1937 by Dr. Paul Mahoney showed the "carina markedly thickened, the right main bronchus filled with granulation tissue and necrotic. The left was partially filled with tissue, but showed no evidence of necrosis." Tissue removed from the left main bronchus on biopsy revealed grade 2 carcinoma. The disease was so extensive that a pneumonectomy was contraindicated.

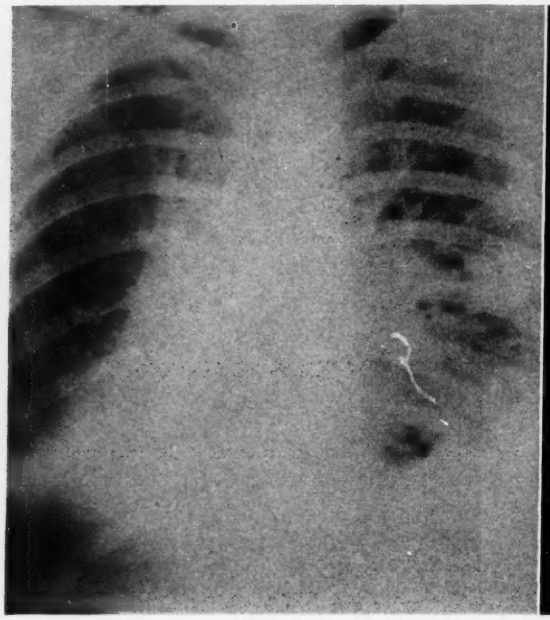

Fig. 2.

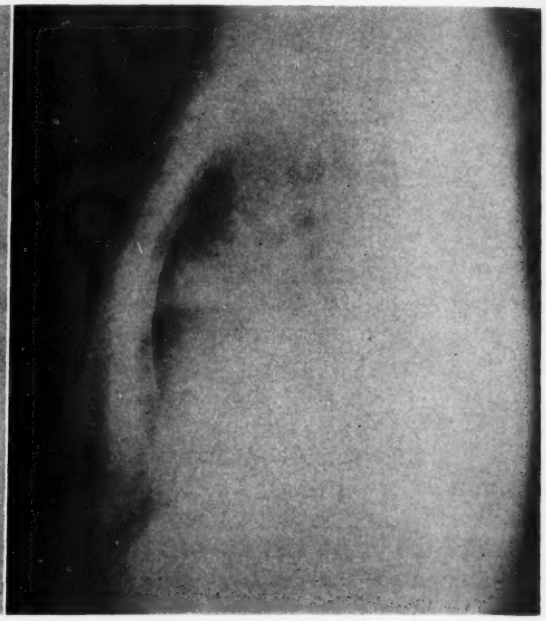

Fig. 3.

Figs. 2 and 3. Case II. The somewhat irregular shadows partially obscured by the heart shadow indicate the area of atelectasis and pneumonitis. These complications followed the blocking of the bronchus by inflammatory and granulation tissue which formed about the aspirated foreign body. Bronchoscopic examination should have been done earlier in this case. Purely symptomatic treatment was continued too long under the diagnosis of lobar or "unresolved" pneumonia.

The patient died on April 3, and autopsy showed carcinoma of right lung, originating probably in the right main bronchus.

Case II. C. G., a white female, aged 43 years, was admitted September 10, 1937, complaining of general weakness, cough, moderately profuse and odoriferous expectoration of thick, yellow, purulent sputum, high fever, and pain in the left chest. She had been in good health until about four weeks previously, at which time she had an upper respiratory syndrome which she believed was caused by pneumonia.

Her temperature on admittance at 12 noon was 104 degrees and at 12 midnight 100.5. The following day a tentative diagnosis of lung abscess of the left lower lobe or tubercular pneumonia was made, with a third possibility of fungus infection of lung. (Figs. 2 and 3.) A guarded prognosis was offered. On September 13, the general condition appeared essentially the same, but the diagnosis of lobar pneumonia in the left lower lobe and beginning pneumonia in the right was given. Active tuberculosis possibly underlay the pneumonia. A week later the temperature was running a septic course, and the patient's condition seemed to be slowly becoming worse. The urine on September 22 showed acid-fast organisms and guinea-pig inoculation was ordered.

The chest surgery division was asked to see this patient on the following day. As far as we could determine from the patient and the intern, the amount of sputum in twenty-four hours was from 200 to 300 c.c., having perhaps diminished slightly in amount during the past week. The sputum was slightly greenish and thick, but not particularly odoriferous. No separation into layers occurred upon standing. The history of good health up to about eight weeks before; the onset of a "cold," with no aching of the bones but some semiproductive cough, loss of pep and malaise; the gradual approach of the acute stage, with high septic temperature, productive purulent cough, some pain in the left chest low down; the respira-

tion, general appearance, and the laboratory findings indicated several possibilities probably not of pneumococcic etiology. Tuberculosis might possibly have been present, but we doubted its importance as an immediate therapeutic or etiologic concern. The markedly increased breath and voice sounds over the greater part of the left lung base region posteriorly, the diminution in resonance but lack of marked dulness, and the history suggested pyogenic involvement of the left lower lobe in the region of the abnormal physical and roentgenologic findings, with possibilities of other underlying pathology associated with the pyogenic involvement. There was no history of aspiration of a foreign body, nor any factor suggestive of embolism. The history would cast a doubt upon any pulmonary plugging from bronchial secretions playing a prominent initial part in an atelectasis. An atelectatic phenomenon, however, associated with plugging secondary to possible aspiration, bacterial and mucous collection, or associated with new growth could not be ruled out.

The immediate consideration of practical importance appeared to be the lower lobe involvement of pyogenic nature which had assumed the importance of a lung abscess. The indicated diagnostic procedure was clear: a bronchoscopic examination was definitely necessary, to be followed, if indicated, by the injection of iodized oil. Good postural drainage would possibly be increased by this procedure. At the same time further useful diagnostic findings might present themselves through the bronchoscopic examination. Should a new growth or other visible complicating pathology appear to complicate the pyogenic picture the therapeutic considerations would, of course, be partly cleared. In the event that the case was one of uncomplicated abscess, conservative treatment, with or without repeated bronchoscopic drainage, could be pursued until and if surgery became indicated.

Following this consultation no immediate change in treatment was made, the attendants having decided on further delay with additional observation. From September 28 to October 7 little change occurred, but on the latter date a change of service occurred. The new attendant diagnosed "unresolved lobar pneumonia" of the left lower lobe.

One week later he ordered a bronchoscopic examination. *A small piece of wooden stick about ¼ inch long was removed from the suppurating granulation tissue in the left main bronchus.* It was then found that the patient had a habit of chewing sticks and matches. Following procurement of drainage by removal of this foreign body and the granulation tissue about it, the patient entered immediately into an uneventful recovery.

Case III. L. D., a white male, 17 years old, was admitted April 16, 1937 with a history of "pneumonia." This had begun about 6 weeks previously, but he had never recovered. There had been a loss of 25 pounds in weight. All signs and symptoms pointed to massive empyema (Fig. 4.)

Recommendation had been made that drainage tubes be inserted through small thoracotomy openings. The day after admission the chest was aspirated in a right interspace of the mid-axillary line by the resident surgeon. No pus was obtained. Aspiration was then done at the angle of the scapula and a few c.c. of thick, creamy, greenish pus was obtained. Another tap the next day was unsuccessful, and the possibility of encapsulated pockets was mentioned. On April 19, ½ to 1 ounce of purulent fluid was aspirated in different areas, indicating the presence of pockets. Exploratory thoracotomy was indicated.

On April 20 about 5 cm. of each of the eighth and ninth ribs was resected in the posterior axillary line. The parietal pleura was opened and several different, firmly encapsulated pockets of pus were found in the right pleural cavity. There was one large interlobar encapsulation of pus between the middle and lower lobe which contained 8 ounces or more of clotted purulent odoriferous exudate resembling very much in appearance dirty clabber milk. Some of the pockets were filled with dirty grayish pus, others with pus somewhat thicker and whiter. All septa of the separate pockets were broken down and closed drainage was instituted. The patient had a satisfactory recovery without further chest complications and the lung expanded to its full capacity before his discharge on May 20, 1937.

Case IV. H. B., a white male of 62, was admitted January 1, 1938 with a complaint of a dry cough which had begun about one year previously.

Eight months prior to admittance he quit work and was sent to a sanatorium with a diagnosis of tuberculosis without positive sputum. He was then sent to the City Hospital for a bronchoscopic examination, which re-

vealed a rather firm mass in the right main bronchus, approximately 1 inch distal to the primary bifurcation. The bronchus lumen was almost entirely occluded. A biopsy revealed squamous cell carcinoma. (Fig. 5.)

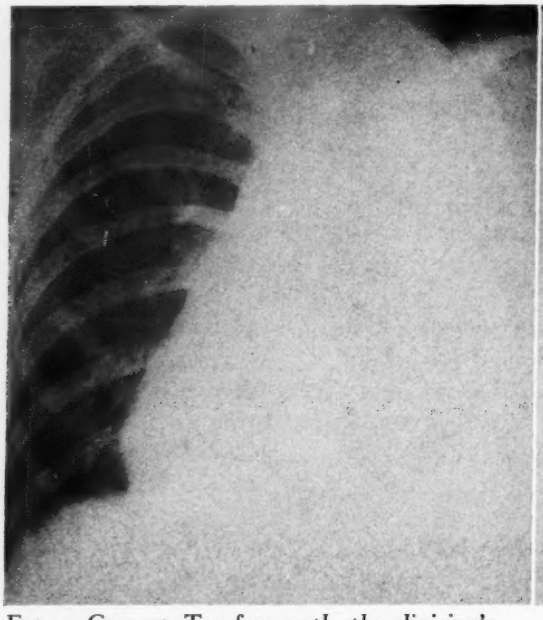

FIG. 4. Case III. Too frequently the clinician's diagnostic curiosity is satisfied with a roentgenologic report of empyema. Though a massive empyema may be present, as is true here, it is always necessary to inquire carefully into the cause. In this case the empyema followed pneumonia. In Case 1 we obtained a similar picture and an *x*-ray report of empyema, but the primary etiologic factor was a bronchial carcinoma.

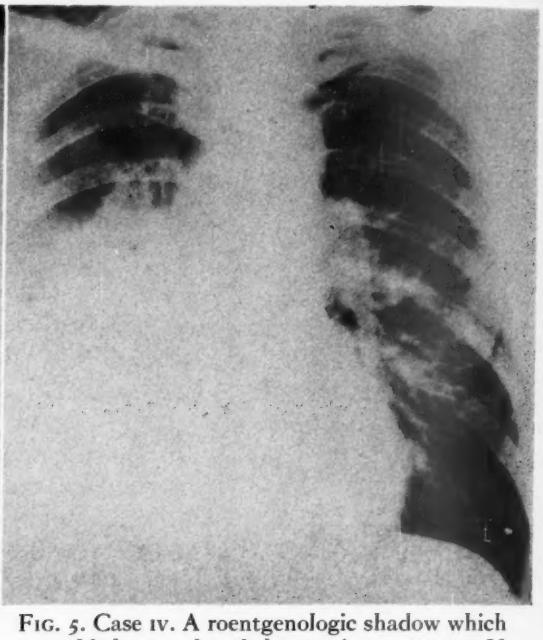

FIG. 5. Case IV. A roentgenologic shadow which could be produced by various causes. If diagnostic curiosity had been satisfied with a diagnosis of lung abscess without inquiry into the specific cause, the bronchogenic carcinoma which was visualized through the bronchoscope and which blocked the lumen of the bronchus giving atelectasis and lung abscess, would probably never have been discovered.

After preliminary pneumothorax, pneumonectomy was attempted on January 14. The right mediastinal pleura was reflected after the method of Rienhoff, and an excellent exposure of the right hilus was obtained. The large primary trunks of the pulmonary veins were rapidly made visible without any untoward effects. At this stage it appeared that individual ligation of the structures of the hilus would be relatively easy. The deep structures of the hilus, however, were thoroughly caked with inflammatory and neoplastic tissue and were so tenaciously buried within this tissue that the arteries and veins could not be isolated without hemorrhage. We were determined, however, to do a clean glandular dissection of the hilus with removal of the lung and hilus at the bifurcation of the trachea. This was the patient's only chance for recovery. This method appeared preferable to mass ligation with removal of the lung, which meant leaving carcinoma throughout an undisturbed hilus. Though the lung was finally removed, a considerable amount of hemorrhage was encountered. The patient was given a blood transfusion on the table, but he died shortly after the lung was extirpated.

The bronchogenic carcinoma was complicated also by a pulmonary abscess.

DISCUSSION

In the first case mentioned several errors in treatment were made, and the disregard of proper diagnostic procedures was noticeable. Internists must appreciate that thoracic surgeons have amply proved that a patient with a lung abscess should usually be treated by surgical means if satisfactory improvement in the abscess proper does not occur relatively soon under such conservative measures as postural drainage, attempts at specific drug therapy or bronchoscopic maneuvers. Most of all, one

must realize that a lung abscess may be produced by a variety of factors; unless it is clear that it is of primary pyogenic origin, *one must always look for the mechanism of origin.* Aspirated foreign bodies, carcinomata originating in the large bronchi and partially or completely obstructing and producing atelectasis, stasis and abscess, are relatively frequent causes.

Another interesting factor in the first case was the fact that organisms belonging to the streptothrix group were obtained in pure culture. This led us to believe that an actinomycotic infection was present. This pure culture was secondary, apparently, to a bronchogenic carcinoma. It may be that different organisms were present in the other pockets of this pleural cavity. The accompanying bacteriologic findings illustrate how easily one can become confused. We could have overlooked the entire trend of the pulmonary events, i.e., bronchogenic carcinoma causing lung abscess and, finally, empyema with multiple encapsulations.

I saw recently in consultation a similar case in which spinach swallowed by the patient came out through the incision remaining after an operation for a lung abscess. It had not been previously appreciated that carcinoma of the esophagus was the original lesion. The lung abscess had occurred secondarily, as a result of bacteria and erosion traveling outside the large bronchi into the lung.

Early Bronchoscopic Examination. In the second case cited above there was a history of onset of the illness *twelve weeks* prior to bronchoscopic examination, with a temperature rising to 104 degrees, medical attendants were prone to refer to the pathology as being lobar or "unresolved" pneumonia. Any pulmonary infection caused by the common pyogenic organisms, regardless of whether ordinary lobar or bronchial pneumonia were at the onset an etiologic agent, which has persisted four weeks or longer, must be looked upon as having perhaps assumed the status of more complicated pathology. An incipient lung abscess may be present, or a complicating empyema. Recognized diagnostic and therapeutic measures should be instituted promptly. Ordinary symptomatic medication and watchful waiting are useless. *"Unresolved pneumonia" when used in such manner as above becomes a vague and loose term of doubtful scientific value.*

In Case II, the simple procedure of bronchoscopy was easily completed and a small piece of stick imbedded in purulent obstructing granulation was removed from the left bronchus. Though the patient then made a rapid and uneventful recovery, proper diagnostic and therapeutic effort had been delayed four weeks longer than necessary. This could easily have meant the difference between a full blown abscess and a simple pneumonitis. Furthermore, even now it is impossible to say that the delay has not predisposed toward a future bronchiectasis of troublesome nature because of damage which may have been done to the involved bronchi.

Multiple Encapsulations in Empyema. The third case cited (and the first in certain aspects) is illustrative of a type we frequently see improperly treated. A most inexcusable error, occasionally made, is to allow an empyema complicating a pneumonia to remain too long undiagnosed. In Case III the diagnosis was made by the attending physician before the patient was admitted to the hospital and four weeks after the onset of the pneumonia. This is a much earlier diagnosis than some.

The point to be stressed here is that *multiple encapsulations of pus were present in these two chests.* Had the recommendation of simple drainage been followed, *only one pocket* would have been drained and the patient's convalescence would have been delayed until other pockets had been suspected and properly treated. The patient would have been subjected to additional operative procedures because the full degree of pathology had not been ascertained or suspected before surgery was instituted. Though Lillienthal, in 1922, called attention to the necessity of doing exploratory thoracotomies in many cases

of empyema, particularly the neglected ones, many cases of multiple encapsulations of empyema undergo simple open

Fig. 6. A patient in whom it was necessary eventually to remove ribs over a chronic empyema cavity to allow collapse of the soft parts. A cure was obtained.

drainage, without effort being made to ascertain if multiple pockets are present. Convalescence may be prolonged for many weeks, and death may even occur, before the attendant realizes that pockets are present which have never been drained. Although one prominent thoracic surgeon disagrees somewhat with this attitude, our experiences uphold Lillienthal's point of view. This does not mean, of course, that exploratory thoracotomy is indicated in every case of empyema, but that it is imperative to keep in mind the possibility of multiple encapsulated pockets.

An exploratory thoracotomy may be done with resection of only a bit more rib than is necessary for simple tube instillation. It is frequently necessary to insert only two fingers through the thoracotomy opening to break down different pockets. However, unless this is done sufficiently early when indicated, thoracoplasty may finally become necessary for the obliteration of an old empyema cavity. The neglected adhesions become so thick and profuse that decortication of the lung and ultimate expansion become impossible, even though the adhesions are freed again and again. This end result in another of our patients is illustrated by an *x*-ray plate showing final recovery after removal of overlying ribs following several less radical attempts at cure.

Necessity of Differentiating between Cough and Other Symptoms Caused by Tuberculosis or Bronchogenic Carcinoma. Since bronchogenic carcinoma is no longer an utterly hopeless disease, it is no longer permissible to delay or avoid indicated procedures. Lipiodol injections and bronchoscopic examinations are necessary in patients presenting unexplained cough, possible blood-tinged sputum, and other signs and symptoms so frequently attributed to tuberculosis, unless one has well-founded scientific reasons for the diagnosis of uncomplicated tuberculosis. A typical *x*-ray picture, typical crepitant râles, and, if possible, a positive sputum should be obtained before the attendant dismisses too long from his diagnostic horizon, carcinoma or other non-tuberculous pulmonary afflictions. About 80 to 90 per cent of primary lung carcinomas seem to originate in the large bronchi and a great many of these may be visualized by the bronchoscope early in their development. Biopsy may very frequently be done without excessive danger or discomfort to the patient. If such neoplasms are diagnosed in the early stage it is possible that expert thoracic surgeons may cure, even at this time, 50 per cent(?) or more of lung (bronchogenic) carcinomas by pneumonectomy. *X*-ray treatments are apparently of no value at present and radium implantations are less desirable than surgery in most cases.

SUMMARY

The above discussion of chest problems and the illustrative case reports are an index to the frequency with which physicians not especially interested in this field

are contributing to mortality and morbidity by their failure to pursue proper diagnostic and therapeutic procedures. These errors are made not only by men who neglect altogether to read medical journals, but frequently by otherwise generally efficient physicians and surgeons as well.

Some of the most common errors in diagnostic and therapeutic procedures which are made by the general profession are as follows:

1. Failure to request bronchoscopic examinations in doubtful cases such as those discussed above. Lack of appreciation of the pathology, physiology, and varying etiology of atelectasis and lung abscess seems in particular to be noteworthy, the term "unresolved pneumonia" being used altogether too frequently.

2. Failure to consider the various potential etiologic factors in empyema, thereby allowing diagnostic curiosity to be too frequently satisfied with an *x*-ray report of empyema is common. It is necessary to realize that lung abscesses from various causes, including lung carcinomata and aspirated foreign bodies, may have been present previous to the empyema.

3. Failure to determine whether or not multiple encapsulations are present in neglected cases of empyema and to treat these pockets by open thoracotomy with breaking of the septa instead of simple insertion of a single drainage tube with consequent drainage of only one pocket.

4. The still too frequent tendency to classify all chronic coughs and accompanying symptoms as tubercular; and allowing individuals to go for weeks or months "awaiting developments" when early pursuit of procedures, such as lipiodol injections and bronchoscopy, might be life-saving, particularly in early bronchogenic carcinoma.

5. Failure to appreciate that thoracic surgeons now have ample statistics proving that pneumonectomy, lobectomy, pneumonotomy and thoracoplastic procedures judiciously used are tremendously valuable in otherwise hopeless chest conditions including, in addition to those disorders mentioned above, far advanced bilateral cavernous pulmonary tuberculosis.

BILATERAL EMPYEMA*

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ARTICLES dealing with the subject of empyema have become gradually limited to a discussion of the methods of treatment, drainage, etc., the pathology, pathogenesis and etiology having been well established. In the hands of competent men, accustomed to treating the disease, the resultant mortality is low and the whole subject is well standardized. One variety of empyema of the pleural cavities, however, remains sufficiently rare to warrant further consideration. This is bilateral empyema, which may be considered unusual when one reflects that many surgeons have never seen it at all, or else find their experience limited to one or two cases in civil practice. This is borne out by a rather cursory examination of the literature, in which practically all the reports are limited to one or two cases each, with the exception of the large series reported by Dunham[1] in 1917, from the U. S. Army Camps. Table 1 gives a list of authors and number of cases reported in the literature since 1905.

The first mention of bilateral empyema was apparently made by Charrier[2] in 1855, who saw many cases following an epidemic of puerperal sepsis. Isolated cases have been reported from time to time since then, and these have been summarized and studied by Hellin in 1905,[3] Fabrikant[4] in 1911 and by Keyes[5] in 1931. Our own experience recently in dealing with such a case has prompted this report.

Keyes in his excellent study of this subject reported three cases from the New York Hospital and collected thirty-eight cases from the literature of the previous ten years. He defined bilateral empyema as "a condition in which pus is found in both pleural cavities at or about the same time." There is usually a common etiologic background, since in most cases the contralateral infection may be regarded as a complication of the original unilateral empyema. The cases reported all seem to be acute, including our own.

Incidence. Estimates as to the frequency of bilateral empyema among all cases of empyema vary from 0.6 per cent to 42 per cent. Keyes[5] reported three instances in 495 cases—0.6 per cent; Geitz[6] reported five out of 588 cases; Lord[7] in Osler's "Modern Medicine" found five in 248 cases—2 per cent; Hellin[3] gives the incidence as 7.7 per cent, collecting 113 out of 1,448 cases. Graham[8] in his "Surgical Diagnosis" states that its incidence "is probably less than 5 per cent"; Da Costa,[9] in his textbook, says "double empyema is an extremely fatal condition. In civil practice I have seldom seen it." Following influenza, especially of the epidemic type, the incidence is very much higher. Stone[10] reported nineteen cases out of 100 in 1918—19 per cent; Dunham[1] found 253 out of 603 empyemas to be bilateral in the U. S. Army camps in 1917—42 per cent.

In children, the incidence of bilateral empyema may be somewhat lower, if one includes the cases in adults following influenza. Holt and Howland,[11] in their textbook, state that in children empyema may be bilateral in about 3 per cent, "oftener in infants." Goldberg and Wexler,[12] who in 1936 reported one case in a child and reviewed the literature to 1910, concluded that the incidence (from the collected cases) was 2.28 per cent. In 1933 Mueller and Mogavero[48] reviewed the literature and reported a total of 312 cases collected up to the time of their article. Since then there have been several sporadic

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cases reported,[12,49,50,51,52] including an interesting case by Stenbuck[51] in which, in addition to a bilateral empyema, there was present a purulent pericarditis which was treated by open drainage.

Sex. Among the cases reported, males predominate over females.

Etiology. Pneumonia is the chief etiologic agent, being responsible for over three-quarters of the cases. Seventy-five per cent of these pneumonias were bronchopneumonias. The bronchopneumonias following the exanthemata gave rise to an especially virulent form with very high mortality. Fabrikant[4] reported 20 per cent of his cases of bilateral empyema to be "primary," although this is rather difficult of conception. More rarely, pyemia, tuberculosis, actinomycosis, surgical operations and puerperal sepsis have been recorded as primary causes. Curtis and Bowman[46] in 1932 reported a case in which bilateral staphylococcic empyema resulted from a carbuncle.

Bacteriology. In approximately 70 per cent of cases the pneumococcus is the causative organism. The hemolytic streptococcus is the usual offender in the remainder of the cases, except for scattering reports of other organisms, such as staphylococci, actinomycosis, B. tuberculosis, etc. More rarely, different organisms have been reported in the two sides (Keyes[5]). In the bilateral empyemas following influenza, the usual percentage of pneumococcus 75 per cent—hemolytic streptococcus 25 per cent was reversed, streptococci being found in 73 per cent and pneumococci in 23 per cent of the cases.

Pathology and Pathogenesis. The pathology is in no way different from that of unilateral empyema. In about 50 per cent of the cases, the double involvement was preceded by a bilateral pneumonia. It is especially prone to occur in the weak, debilitated individual, whose resistance has been markedly diminished by the initial empyema. In those cases in which bilateral empyema followed unilateral pneumonia, most of the writers postulate either metastatic infection or a pneumonic focus so small as to have been overlooked. That this does not occur more often undoubtedly is due to rapid formation of antibodies and

Table 1
Cases reported in the literature since 1905

Author	Year	No. Cases Reported
Hellin[3]	1905 1907	113
Fabrikant[4]	1911	118
Edmund[13]	1910	1
Corner and Grant[14]	1911	1
Gand and Poissonnier[15]	1911	1
Bozzetti[16]	1913	1
Zingher[17]	1913	1
MacKenzie[18]	1914	4
Bunts[19]	1914	1
Lund and Morrison[20]	1916	1
Cafritz[21]	1918	1
Norrlin[22]	1919	1
Gundrum[23]	1920	1
Durham[24]	1920	1
Glenn[25]	1920	2
Jehn[26]	1921	2
Ladd and Cutler[27]	1921	3
Stenius[28]	1920	1
Beck[29]	1921	1
Gomez[30]	1921	1
Schweizer[31]	1921	1
Andrenelli[32]	1923	1
Ormos[33]	1924	1
Auer[34]	1925	1
Hedblom[35]	1925	1
Mackey[36]	1925	1
Matthews[37]	1927	1
Graves[38]	1928	1
Scanlan[39]	1928	1
Dodds[40]	1928	2
Tixier and De Seze[41]	1928	1
Ravnitsky and Bogin[42]	1930	5
Manson[43]	1930	1
Keyes[5]	1931	3
Macera[44]	1931	2
Guerin[45]	1932	1
Curtis and Bowman[46]	1932	1
Steinke[47]	1933	8
Mueller and Mogavero[48]	1933	1
D'Arcy McCrea[49]	1934	1
Goldberg and Wexler[12]	1936	1
Harrington, Dorsey, and Strohl[50]	1935	1
Stenbuck[51]	1936	1
Willis[52]	1938	1
Snow[53]	1935	1
Post influenzal cases		
Stone[10]	1918	19
Dunham[1]	1917	253
Total cases reported	....	455

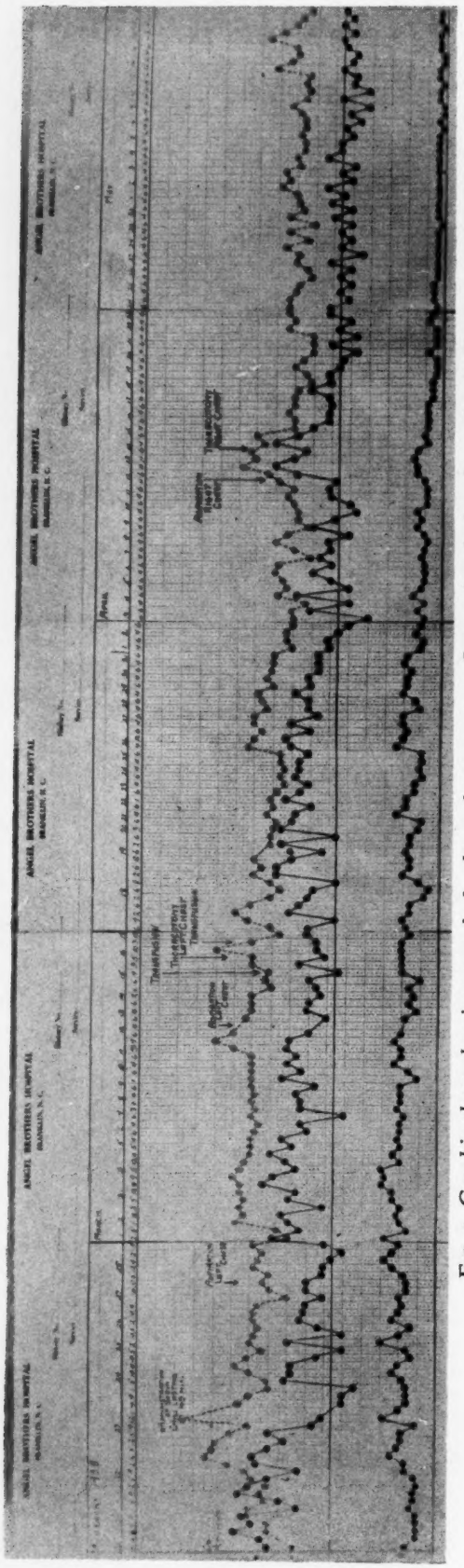

FIG. 1. Graphic chart showing record of pulse and temperature fluctuation during the patient's illness.

local tissue immunity in the majority of cases. In all these cases of double involvement, there must have occurred some

FIG. 2. Radiograph taken during the first week of illness, showing the bilateral pneumonic involvement, further advanced on the left.

interference with the immunity process, a condition which may follow either increased virulence of the invading organism or decreased resistance of the host, or both. Increased virulence of the causative organism was undoubtedly responsible not only for the high incidence of bilateral empyema in the influenza pandemic but for the high mortality as well.

While the time of appearance of the two empyemas may be simultaneous, in the majority of cases one side precedes the other by a few days to several weeks.

Prognosis. The mortality as reported by the various authors varies from 19 to 44 per cent. The lower mortality rates are obtained when dealing with the pneumococcic type, while the mortality from the streptococcic type is exceedingly high.

Treatment. In general the treatment is the same as for unilateral empyema. If the bilateral involvement comes on simultaneously, it is probably better to confine one's efforts to aspiration of the pus as long as possible until the mediastinum has

had time to become relatively fixed by adhesions or induration. In the streptococcic infections, this is notoriously slow in development, usually requiring two to three weeks. Fortunately, in these cases the pus remains thin and is more easily aspirated. When thick pus is present, so that aspiration is difficult or impossible, little is gained by delay and thoracotomy with closed drainage, maintaining a negative intrapleural pressure, should be instituted.

Keyes[5] has ably discussed the question of simultaneous bilateral thoracotomy and concluded that the fears expressed by the earlier writers as to the attendant hazards were unwarranted. He found that "a total of 53 simultaneous or nearly simultaneous thoracotomies had been performed without a single operative death, and with eventual recovery of all but one patient . . . all the above . . . were performed under the 'open' method, . . . less favorable . . . than the safer closed method. . . . Delay in opening the second side, therefore, seems to accomplish little, but to weaken the powers of resistance."

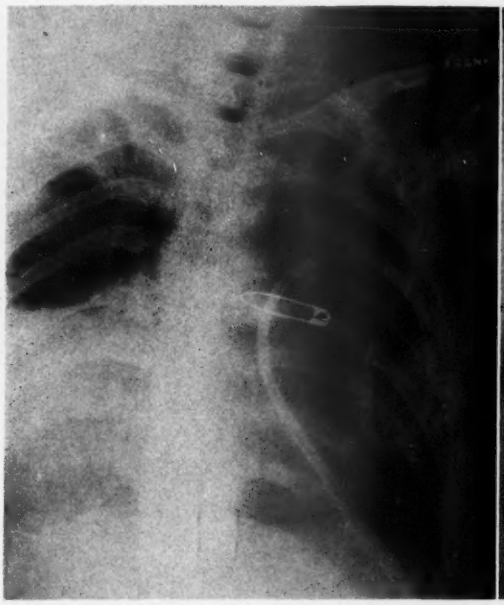

Fig. 3. Radiograph taken April 14, 1938, revealing well-developed empyema on right side, and drainage tube in situ on left. (Unfortunately, the film showing the left-sided empyema before drainage was damaged too badly to be reproduced.)

Fig. 4. Radiograph made May 15, 1938, showing both sides of the chest clearing rapidly.

CASE REPORT

D. S., male, age 38, a forester, was admitted to Angel Hospital February 20, 1938. Chief complaints were stabbing pain in the left chest, exaggerated on breathing, fever and weakness. Three days previously he had developed a cold and cough to which he had paid scant attention. Twenty-fours before admission he had done some rather strenuous labor, becoming overheated and subsequently chilled. Shortly thereafter he had developed fever, malaise, and sharp stabbing pains in the left chest, exaggerated by respirations. He continued to have a slight unproductive cough.

Physical examination revealed a strongly developed adult male, with some dyspnea and slight cyanosis, requiring elevation of the head of the bed for comfort. Temperature was 102°F., pulse 100, respirations 36. There was playing of the ala nasi. Examination of the chest revealed a slight pleural rub, heard over the seventh rib, midaxillary line, and slight impairment to percussion over the left lower chest posteriorly. The heart sounds were normal, the rate rapid but regular. The next morning, there was dulness to percussion over the left base posteriorly and in the midaxillary line, with harsh bronchial breathing. There were numerous coarse râles scattered throughout the chest. The sputum continued scant and grayish in color. It was stained and examined

for microorganisms, but nothing conclusive was found.

An ophthalmic test for serum sensitivity was carried out, and 20,000 units of anti-pneumococcic serum, Types I and II, diluted with 150 c.c. of normal saline solution, were given intravenously, on an empiric basis. Despite the negative ophthalmic test, a violent reaction to the serum ensued, with high fever, very rapid pulse, chill and cyanosis. This required repeated injections of adrenalin and oxygen inhalations by nasal catheter.

Ephedrine was administered in an effort to eliminate any asthmatic factor and to stimulate the circulatory system. Digitalis was administered in order to bolster up the heart.

FIG. 5. Patient shortly before his discharge from the hospital (photographs taken May 15, 1938).

Further inquiry into the patient's history revealed that he was a mild asthmatic, and "wheezed whenever he caught cold." Subsequently coarse moist râles continued scattered throughout the chest, dulness to percussion persisted at the left base posteriorly and he continued to have a high fever and rapid pulse.

On February 27, it was noted that the cyanosis was increasing despite continuous oxygen therapy, the sputum was increasing in amount and there was now absence of breath sounds over the left lung posteriorly, with flatness on percussion, mucous râles continuing over the remainder of the chest. The left chest was tapped in the seventh interspace, posterior axillary line, and 150 c.c. of cloudy yellowish fluid withdrawn. Microscopic examination of this fluid revealed numerous leucocytes but no microorganisms. The pulse rate continued to rise and became somewhat irregular.

On March 8, *x*-ray examination revealed either a beginning lung abscess or an interlobar empyema on the left side. The right lung showed some suspicious mottling but no definite areas of consolidation. At this time the patient's general condition was very poor. He was toxic, disoriented and cyanotic. Inductotherm treatments were given over the left chest, in an effort to encourage localization and resolution of the possible lung abscess. Four days later aspiration of the left chest was carried out in the seventh interspace, posterior axillary line, and cloudy yellowish fluid was obtained. On March 15, pus was obtained by aspiration for the first time very deeply situated, and seemingly directly behind the heart. Because of his desperate condition he was given 350 c.c. of whole blood by direct transfusion in the afternoon and thoracotomy was performed in the patient's room, on the following morning; 1,500 c.c. of thick, yellowish-green pus, without odor, were obtained. Another transfusion of 320 c.c. of whole blood was given the same day.

Irrigation of the cavity with normal saline and Dakin's solution was begun on March 20, and the patient began to improve. However, he continued to run an irregular fever and his pulse rate remained elevated. Coarse mucous

râles persisted over the right chest, he had a productive cough and remained very weak and toxic.

On April 12 flatness on percussion and absent breath sounds were noted over the right base posteriorly. Aspiration revealed cloudy yellowish fluid. *X*-ray two days later revealed empyema of the lower right chest, with displacement of the heart of the left. The left chest was still cloudy, and the drainage tube on the left side was still functioning, copious drainate still going on. Thoracotomy (open) was carried out on the right side the same day and approximately 1,500 c.c. of thick, yellowish-green odorless pus was obtained. From then on, the patient's convalescence was uneventful, albeit slow, and he was finally discharged from the hospital May 24, 1938. At that time the left chest was healed, and the sinus in the right chest was still draining slightly.

DISCUSSION

This case is fairly typical of those already reported in the literature. It is our belief that the severe serum reaction which this patient sustained produced an overwhelming of the immunity process, which led to the ultimate bilateral involvement. This is further borne out by the fact, not mentioned above, that the total leucocyte count on admission was 18,000, while the day following the serum reaction it dropped to 8,000. Also, the patient's asthmatic background undoubtedly interfered with the development of the local tissue immunity necessary to accomplish a successful outcome of the pneumonic process. It may also be worthwhile to point out that both pleurae were draining simultaneously, with open drainage, without any ill effect to the patient, a fact already emphasized by Keyes.[5]

SUMMARY

The incidence, mortality and pathogenesis of bilateral empyema of the pleura are briefly discussed.

A partial review of the literature is appended.

One additional case is reported.

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Overlooked clinically and neglected surgically, the contusion is the most common form of cardiac trauma. The steering-wheel injury is the classic and most common method of infliction.
From—"A Textbook of Surgery" edited by Frederick Christopher (Saunders).

WOUNDS OF THE ESOPHAGUS

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WOUNDS of the esophagus are relatively uncommon, probably because, except when one is eating, the esophagus is a collapsed tube lying well protected behind the trachea and anterior to the vertebral column. This paper excludes those cases of injury resulting from the swallowing of caustic fluids, and those due to penetration of the wall by neoplasms or infection.

Injuries are usually the result of impact by a blunt or sharp object, and may be classed in two general groups, those occurring from within the tube and those from without. In the first group, the swallowing of a tack or an open pin may cause a slight injury, followed by healing without symptoms. Infrequently a sharp object such as a bone or a piece of metal may lacerate the wall, permitting the egress of food and bacteria into the cellular tissues of the neck or mediastinum, with resulting infection. More often a foreign body may lodge in the esophagus, perforating or lacerating the mucosa and causing an inflammation of the wall; with prompt removal the esophagitis rapidly subsides.

Of 205 cases of foreign bodies of various types lodged in the esophagus (reported by Jackson[1]), there were twenty-seven instances of perforation or laceration, with three deaths. In these twenty-seven, seven were in the thoracic portion. Of the three that died, the cervical portion was injured in two and the thoracic in one—all in children. Cellulitis, cervical and mediastinal abscesses and septic pneumonia were listed as the causes of death. Of those that survived the largest number were in children under 5 years, although one patient was 72 years of age.

Because of the type of infecting material and its location, a laceration entirely through the esophageal wall usually results in a cellulitis that runs a fulminating course, causing abscess formation, septic pneumonia and death. The following histories illustrate this type of injury:

Case I. (Weir.[2]) A man of 35, while eating meat, felt a sharp object lodge in the throat. On the fourth day following a swelling developed in the right side of the neck, accompanied by pain and difficulty in swallowing. By the sixth day there was difficulty in breathing; the seventh day, extreme difficulty in swallowing and laryngeal spasms. The temperature was 102.6. Marked edema and cyanosis were present.

Operation. The deep tissues of the neck were infiltrated with pus of fetid odor with several small pus cavities present. The wound was left open and drained. Tracheotomy was later necessary. Death followed on the ninth day.

Post-Mortem Findings. A small quantity of pus was present in the anterior mediastinum. Marked edema of the right side of the epiglottis. Below the cricoid cartilage there were two openings in the esophagus. Pus was present in the intermuscular planes of both sides of the neck.

Case II. (Osgood.[3]) A child of 11 years swallowed a small chicken bone which lodged in the throat. Late the same day soreness of the throat developed. The third day swelling was present on the right side of the neck with marked contraction of the neck muscles. The pulse was rapid and feeble. On the fourth day the same symptoms were present but more severe, with deglutition easy. The sixth day the patient had pneumonia and on the seventh day he died of suffocation.

Post-Mortem Findings. The subcutaneous tissues of the neck contained pus. Pus was present in the anterior mediastinum and in each pleural cavity. There was a left lower lobe pneumonia and a fibrinous pericarditis. On the anterior wall of the esophagus there was a long laceration. Death had resulted from suffocation.

Case III. (Gross.[4]) A sword swallower, while performing his feat, noticed that it hurt him more than usual. He spat up blood immediately. On the third day the pain in the neck was aggravated by swallowing. The neck was swollen and tender with breathing shallow and hurried. The symptoms became more severe and on the fifth day the patient expired.

Post-Mortem Findings. There was a diffuse abscess extending along the trachea and esophagus. The mucous membrane was thickened, tough, smooth and white in color. The outermost coat and surrounding tissues were infiltrated with pus. There was slight excoriation of the upper part with no evidence of perforation or wound. The abscess had been developing prior to the last feat resulting from repeated trauma to the inner lining. The vagus nerve ran through the abscess, causing syncope and death. Edema of the tissues was not sufficient to cause death.

In another group of 628 cases of foreign bodies in the air and food passages reported by Jackson,[5] fifty-three were in the esophagus. One death occurred two years after swallowing a piece of cast iron. The patient had severe inflammation of the esophageal wall and died from bilateral pneumonia.

Apparently the esophagus can withstand most injuries to the mucosa without developing a fatal infection. With laceration of the wall permitting the passage of esophageal contents and particularly infectious agents into the surrounding loose connective tissue, an inflammatory process is initiated, and by muscular action, by gravity, and by the lymphatics, spreads rapidly along fascial planes and establishes itself in various parts of the neck, in the mediastinum, and in the lungs, causing pneumonia. Frequently there develops a suppurative pleurisy and less often a pericarditis.

Because of the nature of the pathologic process and its mode of extension, the mortality rate is very high in complete penetration of the esophageal wall from within outward. This is true in the cases where operation is delayed until the resulting abscesses become localized. Therefore incision and drainage are indicated as soon as esophageal injury can be demonstrated by the esophagoscope. This treatment provides an outlet for the accumulated infectious material and tends to avert its extension along fascial planes. It is reasonable to infer that such measures would materially lower the mortality rate, and it is relatively free from risk.

Injuries of the esophagus from without inward, particularly in the cervical portion, present a more hopeful picture. Of 172 cases that I have found reported in the literature,[6–31] there were sixty-one deaths and 110 recoveries; the result in one case was not stated.

To illustrate this type of injury and because they are reported in detail, the following four cases are selected from the above group:

Case IV. (Nuttall.[22]) A corporal was shot through the neck June 6, 1915. The patient was received in the clearing station twenty-four hours later in collapse and with some dyspnea. He was unable to swallow and he coughed up some blood shortly after being wounded. Treatment was expectant. The third day he was improved but weak. The fifth day there was sudden severe pain in the right thorax, and on the sixth day breath sounds were absent and pleuritic effusion present on the right side. Air hunger increased, the heart failed rapidly and death followed quickly.

Post-Mortem Findings. The bullet entered the middle of the left sternocleidomastoid muscle. It passed between the esophagus and the cervical spine, perforating the posterior esophageal wall and wounded the right internal jugular vein on its posterior surface, then crossed in front of the cervical nerve trunks to come to rest on the scapula. There was a prevertebral abscess and a second larger prevertebral abscess below the former which had broken through the dome of the pleura on its inner aspect into the right pleural cavity. This contained masses of blood clot and a pint of serosanguinous fluid. The tissues of the neck were inflamed.

Whether any surgical procedure could have altered the result in the above case is uncertain. However, drainage provided by

prompt incision and exposure of the bullet tract might have averted the fatal issue.

Case v. (Adams.[29]) In a case of an incised wound, self inflicted, the blade went through the anterior part of the neck, cutting the thyroid and cricoid cartilages, then through the trachea to the fourth ring. There were no important vessels injured. When milk was swallowed it came through the trachea into the wound. The patient was fed by enemata. On the eleventh day, the patient began taking food by mouth. The patient was discharged from the hospital cured on the forty-first day. No operative interference was necessary. The wound was allowed to heal by granulations.

Case vi. (Longmore.[31]) A patient aged 19 years was shot through the neck and immediately spat blood. Drinking water choked him. The patient had difficulty in speaking. Twenty-five days later the patient was well except he had slight difficulty in swallowing and weakness of the voice.

Case vii. (Blood.[30]) A Hindu woman, aged 17 years, was stabbed with a penknife causing a triangular wound just below the angle of the jaw on the right side. The direction of the wound was forward, entering the esophagus just below the thyroid cartilage. A probe passed into the wound easily excited the act of swallowing. Fluids when swallowed appeared in the wound. Treatment was expectant with no attempt at suturing. The wound healed in three days.

The following case recently seen at Knickerbocker Hospital from the surgical service of Dr. C. A. Frink, is added to the above total and reported in detail because of its interest.

A male, aged 10 years, was admitted thirty minutes after a sleighing accident. While descending a hill, he collided with the sleigh immediately in front, the steel runner of which struck him in the right side of the neck. There was an immediate hemorrhage with pain on moving the head.

General examination was negative. There was fresh blood on the posterior pharyngeal wall. At the level of the cricoid cartilage on the right side, there was a transverse laceration about 1 inch long midway between the border of the sternocleidomastoid muscle and the midline of the neck. There was no hemorrhage from the major blood vessels. Coughing and forced expiration caused air to be expelled through the neck wound. The patient was not in shock; pulse was 96, of good quality and regular.

Operation. Two and one-half hours later, under evipal and ether anesthesia, he was operated on. The sternothyroid and the omohyoid muscles were lacerated. No large blood vessels were involved. There was a longitudinal laceration ¾ inch long in the lateral wall of the hypopharynx. A duodenal tube was passed through the esophagus into the stomach without difficulty and a small piece of rubber tissue drain was inserted to the opening in the hypopharynx. The platysma and skin were closed with interrupted silk. On the second day, 20 c.c. of fluid were injected at intervals through the duodenal tube. The temperature was 100.6; pulse 115; respiration 26.

The seventh day the wound was infected, and all sutures were removed. The patient was still being fed by the duodenal tube. On the ninth day pus was draining from the wound with no involvement of the deep tissues. The patient was ambulatory but was still being fed by the tube. By the eleventh day, the patient was eating well and normally. The wound was closing by granulations. On the fourteenth day the wound was closed and the patient was discharged cured. The highest white blood count was 13,100, with 80 per cent polynuclears.

Not improbably the favorable result in this case was due to the prompt establishment of adequate drainage.

Because of their inaccessibility and ease of injuring vital structures, wounds of the esophagus in the thoracic portion are more likely to terminate fatally. In a study of 8,000 enlisted officers and men injured in battle in 1917–1918,[19] four were listed as injuries to the esophagus in this region, with three deaths. Neither the causes of death or the treatment adopted were stated.

Any attempt to suture a laceration of the esophagus is, I believe, harmful. Its structure of thin muscle lined with mucous membrane is friable tissue in which, even in the most accessible areas, it would be difficult to have the sutures hold. Furthermore, the presence of bacteria in the depths

of the wound and the extravasated blood in the loose cellular tissue provide an ideal medium for the development of an inflam-

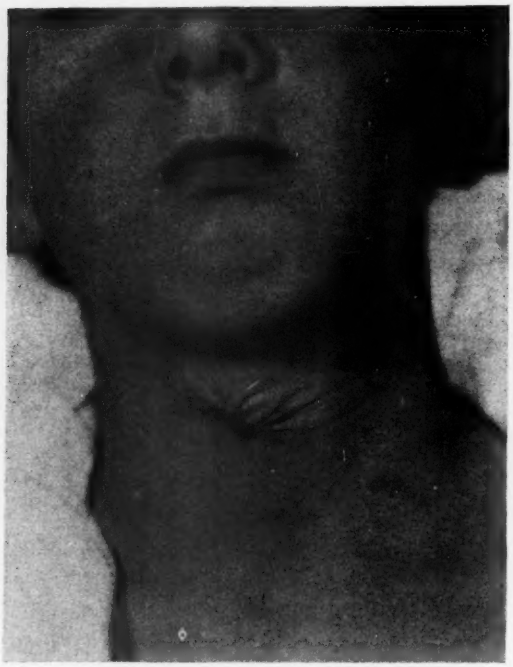

Fig. 1. Appearance of neck wound eight months after injury.

matory process and a cellulitis which extends rapidly. The added exposure necessary to suture the esophageal wall would open up new avenues and thereby favor the spread of infection. The sound principles responsible for the evolvement of the two stage operation for diverticulum of the esophagus, lessening the probability of a fatal infection, apply equally in the treatment of these injuries. The wound should be left open, and lightly packed to permit adequate drainage; food and water should be given through an esophageal tube.

The advice of Adams eighty-five years ago, emphasizing the open treatment of such injuries, holds good today. In reporting his case, he concluded his remarks with: "Nor should it be passed unnoticed how well was exemplified in the foregoing case the propriety of avoiding plaster and sutures as is generally advised by systematic writers when treating wounds of the throat."

The frequency of cicatricial stenosis following esophageal injuries has been the subject of considerable discussion. Injuries occurring from within outward result in stenosis in a higher percentage of cases than

Fig. 2. *X*-ray of hypopharynx and esophagus. No evidence of stricture.

do those from without inward. Probably this is the result of the associated inflammation of the esophageal wall itself.

In the second group, if any of the laryngeal cartilages have been injured, even though the injury is limited to the perichondrium, there is usually a perichondritis and abscess with chondral necrosis and laryngeal stenosis. Jackson[32] states, "Chronic stenosis of the hypopharynx or of the cervical esophagus occurs in at least half of the cases."

In the case here reported a study was made eight months after injury; roentgenologic examination showed no evidence of narrowing or irregularity. (Figs. 1 and 2.)

CONCLUSIONS

1. The esophagus apparently withstands superficial injuries to a remarkable degree without developing serious infection.

2. A laceration entirely through the wall, from within outward, results in a fulminating infection of the cellular tissues of the neck and mediastinum, and, frequently, suppurative pleuritis, pneumonia and pericarditis. The mortality rate is high.

3. Because of this high mortality, early surgical drainage is indicated to avert, as

far as possible, the spreading of the infection.

4. In injuries from without inward, early establishment of drainage is indicated with no attempt at suture of the deep structures.

5. All bleeding from vessels should be promptly controlled by ligature. Feeding should be done through an esophageal tube for a period of ten days to two weeks.

6. Stenosis of the hypopharynx is a relatively common occurrence following injury.

7. Early diagnosis and early surgical drainage tend to reduce mortality and the complications which result in stenosis.

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THE FALLIBILITY OF PROLAN A EXCRETION AS A PROGNOSTIC AGENT IN CASES OF TERATOMA TESTIS

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DURING the last decade biochemical research on the sex hormones has added materially to our clinical knowledge. At the same time clinical observations have led to further study. Since Zondek first noted the appearance of the sex hormone of the anterior hypophysis, prolan A, in the urine of a man suffering from teratoma testis, numerous observers in many countries have made similar observations, and Ferguson has pointed out that the appearance of this hormone, which is not normally present in the urine, constitutes a valuable diagnostic agent. The writer's experience confirms this point of view. Prolan A is not present in the urine of healthy men, nor in that of patients with benign lesions of the testis; but it is consistently present in those with teratoma testis.

Its value as a prognostic agent is less certain. The case cited here is reported with the hope that it may throw further light on this questionable point. In 1934 Ferguson[1] reported the effects of irradiation treatment on the prolan A output. He noted that if the excretion of the hormone was unaffected by irradiation, the prognosis was bad. In all such instances, the patients died. If, however, irradiation treatment was followed by a drop in the excretion of prolan A in the urine, Ferguson considered the rapidity and extent of this drop "a good index of the radiosensitivity of the tumor, and a *reliable* factor on which to base the prognosis." (The italics are ours.) He further observes that in all of the recurrent cases in his series, "an increase in the excretion of prolan A in the urine was observed anywhere from two weeks to three months before the metastatic lesions became clinically demonstrable."

Our experience with the following case in which surgical removal of the tumor was followed by a drop in prolan A output from 719 units to 450 units does not lead us to believe that prolan A output is a "reliable" factor on which to base prognosis.

The patient was a male, aged 33 years. His past history was negative except that his aunt had died of cancer of the breast. In March, 1934, during an examination for diabetes mellitus and overweight, the patient remarked that he thought the right testicle was getting larger. The patient was overweight about 80 pounds (weight 240 pounds). The testicle was hard, smooth, symmetrical, and not painful. It seemed probable that if any tumor was present, it was benign; but nevertheless a consultation was held by Drs. Ewing, Ferguson, and the writer. An aspiration biopsy was advised and the condition proved to be teratoma, grade 4, of the right testicle.

X-ray therapy to the testicle, abdomen, and chest was started immediately. Three months later, June 6, 1934, the tumor had diminished greatly in size and it seemed probable that all the viable cells in the tumor had been destroyed. The testicle and cord with all its contents were removed surgically. Pathologic studies of the tissue from the cord and primary tumor showed no viable tumor cells.

Estimates of the prolan A excretion in the urine were begun on April 30, 1934, one month after recognition of the tumor. At this time there were 2,000 units; three days later the output had dropped to 250 units under irradiation treatment, and we became hopeful of a good prognosis, basing our judgment on the rapid drop. On May 14, there was a rise to 1,000 units, but after eight more days, the output had again dropped to 200 units. Operation was performed on June 6. On the 14th, the output was 350 units, but as Ferguson had stated that an output of 400 mouse units or less was consistent with clinical cure, we were

still hopeful. *X*-ray examination of the chest was negative, and by April 8, 1935 the prolan A output had dropped to 100 units. Following this, there was a gradual rise in output to 400 units in September and 500 in December. On December 23, 1935 the patient developed a cough with hemorrhage. The *x*-ray showed a large, single, metastatic nodule at the right base, just above the diaphragm. *X*-ray therapy was given to this nodule, and the prolan A output again dropped rapidly, reaching 100 units in April, 1936. But the patient's condition grew steadily worse. He expired on October 31, 1936. The last prolan A reading was 500 units on July 27, 1936.

This case is interesting because of the high degree of fluctuation in the prolan A output, with the rapid declines to as low as 100 units, giving us hope that the patient would escape metastasis and recover. This may be regarded as an exceptional case. However, as we have no way of knowing when we are dealing with an exceptional case, it seems inadvisable to place too great reliance on prolan A output as a prognostic agent. Although there was a gradual rise in output just before the appearance of metastasis, this was not so marked as Ferguson's observations would have led us to believe.

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Hormones do not act in vitro on inanimate matter. Their metabolic effects are obtained only by contact with living cells.
From—"The Endocrine Glands" by Max A. Goldzieher (D. Appleton-Century).

TUBERCULOUS MENINGITIS FOLLOWING NEPHRECTOMY FOR RENAL TUBERCULOSIS*†

THREE CASE REPORTS

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ALTHOUGH there have been quite a number of cases of tuberculous meningitis following nephrectomy for renal tuberculosis reported in the literature, yet this sequel is of sufficient rarity to justify recording these cases in detail. In searching through the cases of the Department of Urology (James Buchanan Brady Foundation) of the New York Hospital, only three such records have been found. It is most likely that the meningitis would have developed in these cases, irrespective of nephrectomy, as a result of tuberculous lesions elsewhere in the body. It should be stressed that, in the local treatment of renal tuberculosis, one should be guided by the general physical condition of the entire body with reference to tuberculous lesions existing in other organs before local measures are instituted.

CASE 1. C. E., age 23, single, was admitted to the Department of Urology (James Buchanan Brady Foundation) on October 15, 1935, complaining of frequency, urgency, burning, and nocturia. Approximately one year before admission he was accidentally struck on his right testicle, which soon became swollen and painful. The pain subsided within a week, but the swelling persisted. Two months after this accident he began to have burning on urination, urgency, frequency and nocturia, followed within two months by a painless hematuria associated with the passage of gross particles of "flesh." Five months after the testicle became swollen a right orchidectomy was performed in another hospital, which returned a pathologic report of tuberculous epididymitis. The bladder symptoms of frequency, urgency, burning, and nocturia gradually became exaggerated, so that he was voiding every half hour during the day, and with considerable discomfort.

The patient had enjoyed good health until his present illness began. He had bronchopneumonia without sequelae at the age of 10 years and rheumatic fever without sequelae as a child. The only operation he had experienced was the right orchidectomy, seven months before admission.

Family history was irrelevant, except that one paternal aunt had tuberculosis, but the patient had not been exposed.

Physical Examination. The patient was a fairly well developed and well nourished white male of 23 years, in no acute distress, alert and coöperative. There was no local or general glandular enlargement. Examination of the head, neck, and chest revealed no pathologic changes except dental caries, and granular breath sounds, but no râles. The right kidney was easily palpable, but not tender. The penis was normal; the right testicle absent; the left epididymis was indurated and enlarged at the globus major. The prostate was normal in size, elastic in consistency, non-tender, and the seminal vesicles were not palpable. Reflexes were present and active.

The urine was clear and acid, showed a trace of albumin, but was negative for sugar. An occasional clump of leucocytes but no red blood cells were demonstrable. The blood Wassermann was negative. Blood urea was 8 mg. per 100 c.c., and the blood sugar 111 mg. Hemoglobin was 90 per cent; red blood cells 4,800,000; white cells 8,000 with 63 per cent polys. The sediment from centrifuged urine, stained with Ziehl-Neelsen stain, was positive for acid-fast bacilli and the guinea pig inoculation test was positive.

* From the Department of Urology (James Buchanan Brady Foundation) of the New York Hospital. These cases are reported with the permission of Dr. O. S. Lowsley, Director of the Brady Foundation, to whom the author is most grateful.

† Presented before the Section of Genito-Urinary Surgery, New York Academy of Medicine, January 19, 1938.

Cystoscopy was done under spinal anesthesia, the No. 24 F. Brown Buerger cystoscope passing without difficulty. The vesical fundus showed generalized edema with an unusual amount of edema and injection in the region of the right ureteral orifice. The left ureteral orifice was also edematous and injected, but not so markedly as the right. The vesical orifice was granular and bled easily upon manipulation of the cystoscope. A No. 5 F. lead catheter passed to the kidney pelvis on the right side with great difficulty, while one of this caliber passed to the left kidney pelvis with ease. Specimens collected from the kidney pelves showed diminished function of the right kidney.

X-rays showed the kidney shadows to be normal in size and in good position. There were no areas of calcification in the urinary tract. The right pyelogram showed shaggy, distorted and distended middle and lower calyces, while the upper calyx was normal. The right ureterogram had a beaded appearance indicative of disease in the upper portion of this organ. The left pyeloureterogram showed a bifid type of kidney pelvis, with normal calyces. The left ureter presented no pathologic changes. A diagnosis of tuberculosis of the lower pole of the right kidney and ureter was made. (Fig. 1.)

X-ray of the chest did not suggest recent pulmonary involvement.

Operation. On October 18, 1935, a right nephrectomy was done by Dr. O. S. Lowsley under spinal anesthesia, using 200 mg. of novocaine crystals. The lower pole of the kidney was densely adherent, but was delivered into the wound without difficulty. The ureter was dissected down as low as possible, ligated and severed, phenol and glycerin being used to cauterize the stump. A kidney pedicle clamp was placed on the pedicle, and two No. 2 ligatures were placed below this. The kidney was then cut off above the pedicle clamp, leaving a good rosette of tissue, and following this a transfixation suture was placed through the renal pedicle. The wound was repaired in the usual manner, with one Penrose drain. The patient left the operating room in excellent condition.

Convalescence. For the first six days postoperatively the temperature ranged between 102 and 104 degrees, with a pulse from 90 to 100 per minute. The wound drained a moderate amount of purulent material. There was some abdominal distention which disappeared on the fifth postoperative day. His fluid intake was fairly satisfactory and there was no evidence of

Fig. 1. Case 1. Note shaggy, moth-eaten, distorted and distended middle and lower calyces on the right side. Also the right ureter is slightly dilated with a beaded appearance. The left pyeloureterogram is normal.

deficiency of the left kidney. At no time during his postoperative hospital convalescence did he complain of headache.

The patient was discharged on November 10, 1935, the twenty-third postoperative day, feeling quite well except for the symptoms of cystitis.

The pathologist described the kidney as follows: The upper two-thirds of the kidney were grossly normal in appearance except for a slight dilatation of the pelvis. The lower third was partially destroyed by an ulcerative process involving the calyces, and also decreased in size due to a depressed scar of the lateral aspect. The cavities were lined by soft yellowish-white tissue, in places distinctly caseous in appearance. The microscopic examination was typical of renal tuberculosis, showing epithelioid cells and tubercles.

Exactly forty days after dismissal from the hospital, December 20, 1935, the patient was readmitted with the following history. After returning home he remained on a limited routine with rest and gradually regained

strength. Approximately two weeks before this admission he developed a "cold" in his head and chest, accompanied by chills, fever, and dull, aching pains in his legs. With the continuance of the chills and fever, the pain seemed to progress upward in his body and spine to his shoulders, neck, and head. At the time of admission he complained of severe pain and stiffness of his neck. Nervousness was a prominent feature, requiring sedatives for rest.

The positive physical findings at this time were: slight strabismus of left eye, discharge from right ear drum, stiffness of the neck, well healed right nephrectomy scar, a positive Kernig, and hyperactive reflexes. The white cell count was 10,000, with 79 per cent polys.

On December 22, 1935, 65 days after the nephrectomy, a spinal puncture was done. The spinal fluid was under considerable pressure and cloudy, and the cell count was 509 (lymphocytes), but no tubercle bacilli or other bacteria could be demonstrated on smears. A culture, however, was positive for tubercle bacilli after thirty-five days. The temperature fluctuated between 99 and 101 degrees. Frequent spinal punctures were done to relieve the pressure and he was treated with old tuberculin. He gradually became mentally confused, irrational, comatose, and died on January 10, 1936, twenty-one days after the second admission and eighty-four days postoperatively.

A complete autopsy was obtained, and the following diagnoses were made: (1) tuberculosis of stump of right ureter; (2) tuberculosis of urinary bladder, miliary tubercles of spleen and liver, calcified tuberculous nodules of bronchial lymph node on the left, and bilateral otitis media. The neurologic examination revealed a marked meningeal exudate covering the base of the brain with a lesser amount of exudate over the hemispheres. Marked congestion of the cortical veins was present. There was a beginning hydrocephalus of the lateral ventricles from obstruction by the exudate at the base of the brain. The microscopic picture was one of tuberculous meningitis.

Case II. Mrs. M. M., age 43, was admitted to the Department of Urology, James Buchanan Brady Foundation, on December 6, 1936, complaining of pus in her urine. Approximately two months before admission she noticed that her urine was cloudy. She had no dysuria, burning, frequency, urgency, nocturia, or hematuria. She had her urine examined and it showed numerous pus cells. Two years before admission had had an attack of dysuria, frequency, and nocturia which lasted several weeks, and then subsided after oral medication. Three months before admission she reported to her local physician because of loss of weight and a feeling of malaise. Since she failed to improve she was referred for further study.

The patient had developed pleurisy ten years before, and one year later a diagnosis of pulmonary tuberculosis was made. She was treated by rest, diet, sunshine, etc., for a period of one year, and was told she was cured. Up to three months before the present admission she had felt quite well, with no clinical symptoms of pulmonary tuberculosis.

Family history was entirely irrelevant.

Physical examination revealed a fairly well developed and nourished middle aged female in no acute distress. The head, neck, and chest showed no pathologic changes, there being no râles or abnormal dull areas present. The abdomen was normal, the lower pole of the left kidney being palpable but not tender. The rest of the examination was normal, except for slightly hyperactive reflexes.

Urinalysis showed the urine to be cloudy, acid, with a specific gravity of 1.020, a trace of albumin, no sugar, 5 to 10 red blood cells and numerous pus cells to the high power field. Urinary sediment stained with carbolfuchsin showed a moderate number of acid-fast bacilli. Hemoglobin was 78 per cent, and white blood cells 8,800. The Wassermann was negative. The blood urea was 10 mg., and the blood sugar 90 mg. per 100 c.c. of blood.

Cystoscopy and Pyelograms. The No. 24 F. Brown Buerger cystoscope passed easily. The bladder showed generalized cystitis with marked trigonitis. The left ureteral orifice could not be visualized owing to inflammation and edema, and the right ureteral orifice was slightly injected. A No. 6 F. catheter was passed to the left kidney pelvis without obstruction, but a similar catheter passed only one half the distance to the right kidney pelvis, meeting an obstruction at this point. Specimens collected showed a diminution in function of the left kidney, the phenolsulphonphthalein appearing in ten minutes from the left side and 4 per cent being excreted in ten minutes, as compared with an appearance in five minutes with 8 per cent excreted in ten minutes on the right side. There were also 50 red and 50 white cells to

the high power field from the left pelvis, while the urine from the right kidney was free of pus and blood.

X-Rays. The plain plate showed both kidney shadows normal in size and in good position. Near the tip of the left catheter was a shadow approximately 7 mm. in diameter which probably represented a calculus. No other shadows indicative of calculi were noted in the urinary tract. The right pyelogram indicated that the pelvis was bifid in type but within normal limits. The right ureter filled incompletely, but no evidence of pathologic change was noted. The left pyelogram showed the pelvis of normal size, but revealed an absence of the superior and inferior major calyces; those that were present were slightly dilated and blunted. The left ureter filled well throughout, with a suggestion of a slight angulation at the ureteropelvic junction. The remainder of the ureter was slightly dilated and tortuous. A diagnosis of left renal tuberculosis was made and the patient was advised to have a left nephrectomy. (Fig. 2.)

Operation. On December 7, 1936, a left nephrectomy was performed under spinal anesthesia, using 150 mg. of novocaine crystals. The kidney was easily freed from the surrounding fat and removed without difficulty with the same technique as that described in the first case. The patient left the operating room in excellent condition.

Convalescence was entirely uneventful, the temperature not going above 101°F. The patient was discharged on the eighteenth postoperative day, December 25, 1936, with the wound completely healed.

Pathologic Report. The kidney was usual in size, but the capsule stripped with some difficulty in a few areas. There were a few demonstrable tubercles on the surface. On serial section, there were many areas of cheesy granular material, confined mainly to the upper and lower poles. Several of these caseous areas had ulcerated through into the minor calyces. The kidney pelvis was slightly dilated and the mucosal lining thickened with several minute yellow nodules. The left ureter was not abnormal. Microscopic examination revealed many tubercles, with the typical Langhans giant cells.

Three weeks after discharge, or thirty-nine days after operation, she was readmitted to the hospital complaining of frontal headaches, stiffness of the neck and generalized body pains of four days' duration.

She also complained of burning, frequency,

Fig. 2. Case 11. Note absence of superior and inferior calyces, while those that are present are slightly dilated and blunted on the left side. The left ureter is slightly dilated and tortuous. The right pyelogram shows a bifid pelvis.

nocturia, and dysuria. She was nervous, had a stiff neck, dulness and scattered râles at the apex of the right lung anteriorly and posteriorly. A well-healed left nephrectomy scar, and hyperactive reflexes completed the picture. Her temperature was 100.2°F., pulse 90, and respirations 26.

On the day of admission a lumbar puncture was done, removing 15 c.c. of slightly cloudy spinal fluid, under increased pressure. The cell count was 500 white corpuscles, 10 per cent of which were lymphocytes, but no tubercle bacilli could be demonstrated. The sugar content was decreased while the protein was increased. Blood examination showed a hemoglobin of 88 per cent, red cells 4,360,000, leucocytes 13,500, polys 84 per cent, lymphocytes 4 per cent, monocytes 12 per cent. *X*-ray of the chest demonstrated an old healed tuberculous process in both the upper lobes.

A second lumbar puncture was performed two days after this admission, and the centrifugalized sediment, stained with carbolfuchsin, contained acid-fast bacilli. A guinea

pig inoculation with the spinal fluid was reported positive for tubercle bacilli six weeks later.

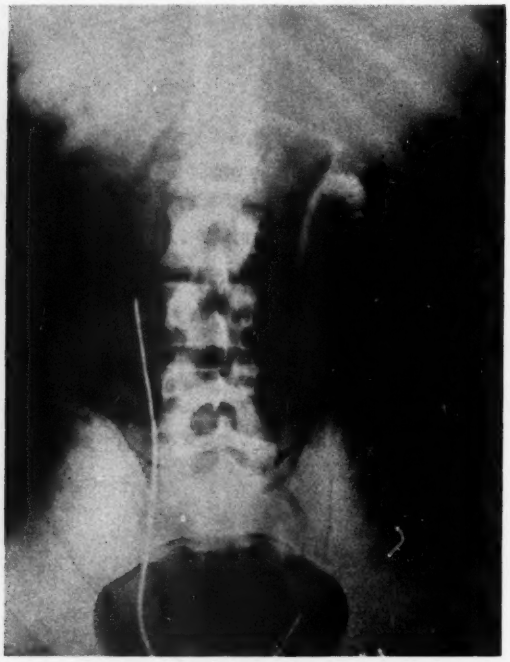

Fig. 3. Case III. The left pyelogram demonstrates a dilated pelvis, the upper calyx being defective in contour with some shagginess, and the middle and lower calyces irregular in outline. The left ureter is dilated and moth-eaten in appearance.

During the two weeks of the second admission, the patient gradually grew progressively worse, having severe headaches, vomiting, rigidity of her neck. She was semicomatose, finally lapsing into coma and expiring on the fifteenth day. After a microscopic diagnosis of tuberculous meningitis was established, treatment consisted of frequent lumbar punctures, sedatives, and nursing care. An autopsy could not be obtained; however, there was no question as to the final diagnosis, since the tubercle bacilli were demonstrated in the spinal fluid as well as by a positive guinea pig inoculation test.

Case III. Mr. F. R., age 28, single, was admitted to the Department of Urology (James Buchanan Brady Foundation) of the New York Hospital on September 29, 1937 complaining of pus and blood in his urine of one month's duration. Associated with these symptoms were slight burning on urination, frequency, and nocturia. Three days before admission he began to have a dull aching pain in the region of his right kidney, associated with an elevation of temperature. His past and family history were irrelevant.

Temperature was 103°F., pulse 108, blood pressure 120/80. The patient was well developed and nourished, but acutely ill. His skin was hot and moist, his lungs showed decreased resonance, bronchial breath sounds, and a few fine râles in the left apex, posteriorly. The heart was normal. The abdomen showed no abnormal masses or tender areas. The genitalia normal and the reflexes present and active.

The urine had a trace of albumin, numerous red blood cells and 1 to 3 leucocytes to the high power field. Urine from the left kidney contained acid-fast bacilli. Hemoglobin was 88 per cent, white count 6,000, blood Wassermann negative. Blood urea was 13 and blood sugar 120 mg. per 100 c.c. of blood. The P.S.P. test gave 96 per cent in three hours. A tryptophane test on the spinal fluid was negative.

Plain *x*-ray plate of the chest showed fairly soft bilateral apical infiltration which suggested recent disease. Cystoscopy indicated good function from both kidneys, but a plain *x*-ray of the abdomen showed a small calculus in the right kidney substance. The left pyelogram showed the upper calyx defective in contour, with some shagginess, and the middle and lower calyces were irregular in outline. The renal pelvis was dilated, and the ureter moderately dilated and moth eaten in appearance. The right pyeloureterogram was normal. Uroselectan series confirmed the retrograde findings. (Fig. 3.)

On October 4, 1937 under spinal anesthesia, a left nephrectomy was done with removal of as much of the ureter as possible. On the day after operation the temperature rose to 104 degrees, and then dropped to 101 on the second postoperative day, gradually returning to normal at the end of the first week. The postoperative convalescence was uneventful except for a slight elevation of temperature and a left frontal headache on the twentieth day, at which time the patient was discharged. A small draining sinus remained in the posterior angle of the nephrectomy wound.

The pathologic report of the kidney and ureter showed renal and ureteral tuberculosis.

The patient was readmitted to the hospital five days after discharge, complaining of a severe frontal headache which had persisted since he had left the hospital. He also complained of slight stiffness of his neck. Except for

this, physical examination at this time was negative. The knee jerks were equal and active, not exaggerated, and Kernig's sign was negative. The blood pressure was 135/85. The left kidney incision was well healed.

A lumbar puncture on the day of admission showed the spinal fluid under increased pressure, clear in color, with a cell count of 55, but with no acid-fast organisms. The tryptophane test was negative. On the second day a bilateral positive Kernig's sign was noted. During the next two weeks, four lumbar punctures were done, which relieved the headache only temporarily. The cell count of each specimen of spinal fluid increased, the last cell count being 320, with 72 per cent lymphocytes. The tryptophane test of the third specimen of spinal fluid was slightly positive for tuberculous meningitis. The patient gradually became moribund and expired on the seventeenth day of the second admission, forty-two days after nephrectomy.

The following diagnoses were made from the autopsy: (1) chronic pulmonary tuberculosis involving the upper fourth of the right upper lobe and upper third of left upper lobe, with fibrosis, caseous and calcified nodules; (2) healed nephrectomy wound (left); (3) acute disseminated miliary tuberculosis with miliary tubercles of all lobes of lungs, tuberculous meningitis (smears made from the meninges showed large numbers of acid-fast organisms), and miliary tubercles in spleen and liver; (4) tuberculosis of left ureter, prostate and right seminal vesicle; (5) chronic cystitis with multiple tuberculous erosions of mucosa; (6) extensive bilateral fibrous pleural adhesions.

CONCLUSIONS

In view of the fact that tuberculous meningitis can follow nephrectomy for renal tuberculosis, one should give a guarded prognosis when patients present symptoms of meningeal irritation after such an operative procedure.

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POSTOPERATIVE CYSTITIS*

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PERHAPS a more fitting title for this paper would be "Postoperative Infections of the Urinary Tract," inasmuch as infection of the bladder by itself does not usually occur.

The diagnosis of "cystitis" should be made advisedly. It is justified only after complete study of the entire urinary apparatus. The clinician's attention is usually drawn to the urinary tract by symptoms arising from the bladder, which may for this reason be regarded as the spokesman for the rest of the urinary system. Inflammatory processes of the kidney, pelvis and ureter have no way of manifesting themselves clinically unless the process is so severe that urinary obstruction occurs, with dilatation of the renal pelvis, or if the inflammatory process involves the capsule of the kidney. These last mentioned conditions will produce renal pain.

The bladder, under normal circumstance, is highly resistant to infection. This statement is supported by abundant evidence, both experimental and clinical. Pathogenic bacteria in large numbers may be introduced into a normal bladder without serious consequence. Infection is seen infrequently even when the introduction of bacteria is accompanied by moderate trauma, as seen following the inexpert or heavy-handed use of the cystoscope. If the above statement were untrue it would be almost impossible to carry on cystoscopy or many of the operative cystoscopic procedures.

Severe postoperative infections are the source of great annoyance to the patient as well as to the surgeon. This complication may prolong the period of convalescence and the distress to the patient may be greater than the operation itself. Fortunately most of the severe types may be either prevented or rapidly cleared up under proper management.

A careful study of postoperative urine specimens will show the presence of leucocytes in a surprising number of cases. Evidence of infection varies from the mildest group, in which pus cells or bacteria are found for a brief period, to the more severe type where infection is evidenced by pus and bacteria in the urine in large quantities and clinical symptoms of chills and fever, dysuria and frequency. In cases of severe systemic reaction as evidenced by chills, high fever and tenderness in the costovertebral areas an infection of the entire urinary tract may be assumed.

Many of the above-mentioned are only laboratory infections, others may be more serious. If treatment is promptly instituted during their incipiency the severe forms may be avoided.

By doing postoperative urine examinations routinely the cause of an otherwise unexplained fever during convalescence may be discovered.

We have pointed out that the bladder may be counted on to stand a great deal of abuse. What then may be considered the predisposing factors in producing the more severe type of postoperative cystitis? Postoperative retention of urine with severe overdistention is the principal factor. The overdistended bladder affords all the necessary prerequisites for infection.

Postoperative distention is produced by an interruption of the reflex arc between the spinal cord and the bladder. This reflex retention occurs following a variety of surgical procedures, more commonly those in the lower part of the abdomen, the inguinal and perineal regions and also after gynecologic surgery and normal labor. It is

* Read before the Surgical Section of the New York Academy of Medicine, April 1, 1938.

probable that the reflex arc is so occupied by the transmission of the pain impulse that the voiding stimulus is unable to make itself felt.

The bladder is a hollow muscular organ lined on the inner side by mucous membrane. On its outer surface large tortuous blood vessels are seen. The wall is capable of a certain degree of stretching and contraction and to that reasonable degree the blood supply has become accustomed. The normal capacity of the bladder is a variable factor, and no doubt many persons may be gradually trained greatly to increase their bladder capacity. If, however, a sudden severe distention occurs there is a stretching of the whole bladder wall which results in a disturbance of its blood supply. The bladder is capable of distention to an enormous degree without rupture; in fact, rupture of the distended bladder from bistention alone is probably unknown.

During the process of distention the thin-walled veins become compressed, the arteries are over-expanded by the positive force of the blood pressure. Following the release of this pressure marked congestion of the mucous membrane of the bladder may occur. Microscopically the picture is one of congestion, edema and petechial hemorrhage. More severe cases show gross bleeding, and the bladder is then properly prepared for infection. This condition has been commonly referred to as a "catheter cystitis." Many patients have been allowed to endure the discomfort of acute retention because the surgeon feared the catheter. If the patient is not permitted to become distended there is no danger in its use. Infection will frequently occur in this damaged mucosa without the introduction of the catheter.

One occasionally hears the remark that the surgeon is reluctant to start catheterizing a patient because he will be obliged to continue it; in other words, that catheterization will delay the resumption of the normal voiding reflex. This is obviously untrue. Certain patients will be delayed for longer periods in reëstablishing their voiding reflex than others, but if the bladder musculature is stretched beyond a certain point it is incapable of forceful contraction and the bladder must be put at rest in order that its musculature may regain its tone.

The management of these cases may be divided into prevention and treatment. Under prevention the most important point is the avoidance of postoperative distention. The question that next occurs is how long shall we let the patient go before using the catheter? Many factors influence the secretion of urine. General anesthetics, particularly ether and nitrous oxide, inhibit its secretion. However, nausea may follow anesthesias so that no fluids will be taken for several hours after. It is difficult to lay down a specific law that will apply to all cases. It is possible to assume however, that normal kidneys will secrete on the average about an ounce of urine per hour. Generally speaking catheterization should be done about twelve hours after operation if the patient has not voided.

If catheterization has been delayed and a large amount of urine has accumulated, it is safer to withdraw only about 12 ounces at a time. The same amount may be withdrawn at two hour intervals until the bladder is emptied. Many observers believe that sudden withdrawal of the entire contents of the bladder adds to the congestion and submucosal hemorrhage. The subject of slow decompression of the bladder is being questioned in certain quarters but so far there are not sufficient data to warrant discontinuing it.

In cases where prolonged postoperative distention has occurred and there is no tendency on the part of the bladder musculature to contract, an indwelling catheter may be employed to advantage. This permits the muscular wall to remain at rest and hastens its recovery. Such indwelling catheters require considerable attention to see that they are draining properly. An obstructed catheter is worse than no catheter at all. The bladder should be washed out thoroughly once or twice a day with a

mild antiseptic solution under strictly aseptic technique.

Several points should be emphasized in connection with the technique of catheterization. This is such a common procedure that one would assume that all the careful detail had been worked out to insure the comfort of the patient. Unfortunately this is not the case. One of the most effective methods of making catheterization less unpleasant is the use of a small catheter; a size 14 F or 16 F is ample. In the female, rigid or semi-rigid catheters of this size are particularly convenient. They may be obtained in woven silk or in a new composition that may be boiled. In the male, a fairly new, soft rubber catheter of the same size is preferred. Old rubber catheters that have been boiled many times lose their firmness and the smooth external covering becomes roughened, making its use difficult. The external meatus is gently sponged with an antiseptic solution. Ample lubrication should be used. (I recently visited a hospital where the use of a lubricant on the catheter was forbidden by the training school.)

It is wise then to place postoperative cases on an eight hour catheterizing schedule if they show no tendency to void in the first ten to twelve hours. As soon as the mechanical difficulties of retention have been taken care of we should further insure ourselves against infection by giving a urinary antiseptic. Fortunately at the present time we have two very efficient drugs for this purpose, namely, ammonium mandelate and sulfanilamide. Ammonium mandelate is probably the less toxic of the two, and it is highly efficient.

Mandelic acid compounds are a direct outgrowth of the use of the ketogenic diet. This diet was too involved to be applied to ambulatory patients, so a search was instituted to find an organic acid that could be taken orally and would be excreted in the urine unchanged. The organic acid found in the almond was the result. It is irritating to the gastric mucosa and its administration has caused considerable gastric distress. Recently it has been put up in enteric-coated tablets. This preparation seems to be very satisfactory.

The bacteriostatic effect of mandelic acid compounds is produced by increasing the acidity of the urine. A hydrogen ion concentration of 5.5 to 5 must be obtained for the best results. The drug should be given in adequate dosage, 4 to 8 gm. a day. It is most efficient when used against the colon bacillus group of organisms which are at fault in about 70 per cent of all urinary infections. Almost all the cases under discussion in this paper fall in this category, with one outstanding exception, the proteus ammoniae or urea-splitting group of organisms. Ammonium mandelate not only does not help these cases, but makes them worse. The ammonium-splitting organisms attack the ammonium mandelate and split off the ammonia; instead of obtaining a higher degree of acidity the opposite is accomplished, and the free ammonia makes the urine less acid.

Administration of mandelate compounds should not be continued in large doses for a period longer than ten days. Prolonged administration is irritating to the kidney and diminished renal function may be demonstrated.

Certain patients are sensitized to mandelic acid. In one case under the author's observation recently, severe edema of the eyelids and lips and ulcerations of the mouth and tongue occurred. Tinnitus and temporary deafness have also been observed. These symptoms clear up rapidly when the drug is discontinued.

Striking results will usually be observed within seventy-two hours. If this does not occur the suspicions of the clinician should become aroused and other studies may be indicated.

Sulfanilamide is an excellent urinary antiseptic and is extremely efficient in doses of as little as 40 gr. a day. The action of sulfanilamide is not definitely established, but it is excreted largely in the urine and is effective when used for all

common bacteria found in the urine with the exception of the staphylococcus.

In contradistinction to the mandelic acid group of antiseptics, the efficiency of sulfanilamide is enhanced by reducing the acidity of the urine. This is readily accomplished by the administration of one dram of bicarbonate of soda or some similar alkali twice daily.

Ordinarily, few if any untoward symptoms will be observed when sulfanilamide is administered in 10 gr. doses, four times daily, for a period of less than ten days. The toxic symptoms produced by this drug occur following the administration of much larger doses.

No urinary antiseptic should be continued for a period of more than ten days. If the urine is not clear by this time adequate investigation is indicated.

If the above-mentioned recommendations are carried out, most of the difficulties may be prevented, although occasionally infections occur in spite of the very best care. The treatment of the well-developed case is in principle the same as that outlined under prevention: catheterize the patient after voiding to be sure he is emptying his bladder. Daily bladder lavage is indicated. Any mild antiseptic solution, for example warm boric solution, may be used for this purpose.

Urinary antiseptics are employed as previously mentioned. Fluid intake is limited to about 2,000 c.c. for each twenty-four hours, so that the antiseptic will not be excessively diluted in the urine. In patients with high fever it is unnecessary to restrict fluids. In certain specialized types of surgery adequate provision for the urinary drainage should be made at the outset. This is particularly true in radical operations on the large bowel where there is considerable disturbance of pelvic structures and injury to the nerve supply to the bladder. Close cooperation between the urologist and the surgeon should be maintained in these cases.

The advent of tidal drainage has been of great benefit in the management of all types of cord bladder during the period of hospitalization. It consists of an indwelling catheter connected with a closed system of irrigation. The irrigating system is equipped with automatic regulators that allow the bladder to be filled and emptied at regular intervals. The rate of filling and emptying may be regulated to almost any speed. Operations on the spinal cord and cauda equina also fall into this group. With this device it is possible to keep such cases free of infection for an indefinite period.

Obstructive lesions should be watched for, particularly in men past 60 years of age. Enlargement of the prostate gland begins to appear at about this time. The patient may be completely unaware that any enlargement of the prostate exists, but following operation, when the voiding stimulus is interfered with, and perhaps the general muscular tone diminished, he finds himself unable to void even after a prolonged period of catheterization. Surgical interference for the relief of obstruction may be necessary.

Another group of postoperative urinary infections does not, strictly speaking, come under the heading of postoperative cystitis, but is mentioned here briefly. This group is composed of those who are obliged to spend a long period of time in bed, following operation, as for example in chronic osteomyelitis, empyema, certain fractures and dislocations immobilized for long periods of time in traction devices, frames, etc.

Any patient confined to bed or immobilized for a long period of time should have regular and careful urine examinations. The urinary tract drains properly only when we are in the erect, normal position. Muscle tone and peristaltic movements of the renal pelvis and ureters are stimulated by our daily activity and better still by regular vigorous exercise. When immobilization of the body is necessary for protracted periods, drainage in the urinary tract is imperfect and chronic infection with calculus formation is not uncommon.

SUMMARY AND CONCLUSIONS

1. Urinary infections in varying degrees of severity are occasionally found following almost any type of surgical procedure.

2. Severe forms of postoperative infection in the urinary tract are usually the result of postoperative distention.

3. Prompt relief of postoperative urinary retention, accompanied by the administration of a urinary antiseptic, will prevent the severe forms of infection. Catheterization should be carried out early.

4. Prolonged periods of bed rest or immobilization of the body by splints, frames, etc., tend to promote urinary stasis. This stasis may result in infection or stone formation.

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THE ANORECTAL ASPECT OF VENEREAL LYMPHOGRANULOMA*†

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AS is well known, up to about five years ago, the general teaching in the United States was that the majority of non-malignant rectal strictures were due to syphilis. The causes of so-called inflammatory rectal strictures, when not attributed to syphilis, were thought to be tuberculous, gonorrheal, or chancroidal infection. Although it is true that occasionally these infections cause rectal stenosis, the experiments, investigations, and reports, here and abroad, in the last twelve years have proved that most of these rectal strictures are manifestations of a protean disease entity, venereal lymphogranuloma.

Historical. Audrey,[1] in 1903, and Puechavy,[1] in 1904, pointed out that the inflammatory rectal strictures which Alfred Fournier[2] described in 1875 and which he named anorectal syphiloma, often accompanied the elephantiasis and progressive ulcerations of the vulva, perineal body, and anal region to which Pierre C. Huguier[3] called attention in 1848 and named esthiomene. In 1913, Durand, Nicolas and Favre[4] fully described the clinical and pathologic features of the inguinal localization of venereal lymphogranuloma. They termed the disease subacute inguinal lymphogranulomatosis and considered it infectious and of venereal origin. In 1925, Frei[5] of Berlin reported a specific intradermal test for this condition. The anorectal aspect of the disease had been considered syphilitic, tuberculous, gonorrheal, chancroidal and even of amebic origin until 1928 when Frei and Koppel[6] discovered that both the inguinal and anorectal aspects of this disease yielded positive reactions to the Frei test. Their observations were confirmed by Jersild[7] in 1930, and by Barthels and Biberstein[8] of Amsterdam in 1931. These findings showed that what primarily had been considered diseased conditions independent of one another were in reality manifestations of a single pathological process of identical etiology.

At the eighth International Congress of Dermatology and Syphilology in Copenhagen, August, 1930, Hellestrom and Wassen[9] announced that they had isolated the virus of this disease. Their investigations were confirmed by Levaditi[10] and his colleagues two months later.

Further studies were carried out and hundreds of reports continued to be published abroad; in 1932 articles began to appear in the American medical literature. The earliest detailed reports in this country were those by De Wolf and Van Cleve,[11] and Sulzberger and Wise.[12] Other articles soon followed, among which were those of Lehmann and Pipkin,[13] Cole,[14] C. F. Martin,[15] Bacon,[16] Bloom,[17] and Grace and Suskind.[18] The enormous amount of literature which has appeared here is proof that the disease is prevalent in the United States, and not confined to the tropics as it was supposed to be. Indeed, it probably occurs with equal frequency in temperate and tropical climates. The real importance of this disease is gradually being recognized in our clinics. The most trying and distressing manifestation is rectal stricture. Chronic ulcerative processes of long stand-

* From the Department of Surgery, Division of Proctology, Brooklyn Hospital (Dr. Ernest K. Tanner, chief attending surgeon). Read before the Section of Surgery, New York Academy of Medicine, December 3, 1937.

† The Frei antigen used in our tests was supplied to us through the courtesy of Drs. H. F. De Wolf, H. N. Cole, C. F. Martin, H. E. Bacon, and B. A. Kornblith.

ing most commonly involving the groin, perineal region, vulva and penis, may also appear.

Race. Venereal lymphogranuloma occurs more frequently among colored peoples than in the white race. This does not signify that negroes are inherently more susceptible to the disease than white people. Factors such as sexual habits must be considered. The expression of the disease in both races, however, is markedly different, both as to severity and variations in types of lesions. This probably is due to what Rosser[19] described as fibroplastic diathesis in the negro race, viz., the tendency in colored people to develop thickening, keloidal formations, and pseudo-elephantiasis in infected and injured tissues. In the white race such tendencies are not pronounced.

Nomenclature: Venereal Lymphogranuloma. Numerous terms have been applied to the various aspects of this condition, the most common of which are lymphogranuloma inguinale,[11] lymphopathia venereum,[12] climatic bubo,[20] tropical bubo,[21] non-tuberculous granulomatous lymphadentitis,[22] anorectal syphiloma,[2] esthiomene,[3] and elephantiasis angiomatosa lymphangiectatica lipomatosa fibrosa.[23] These are but a few of the long array of terms. One cannot help but be impressed by the urgent necessity of some general agreement upon the nomenclature. We favor the term venereal lymphogranuloma which was suggested in a recent editorial of the *Journal of the American Medical Association.*[24] If universally adopted, this name would avoid confusion, designate the venereal origin of the disease, give some conception of the nature of the condition, and embrace the inguinal, anorectal, vulval, penile, buccal and submaxillary localizations and aspects of the disease.

Clinical Features of the Anorectal Aspect. Venereal lymphogranuloma is a disease acquired by sexual intercourse and probably caused by an ultramicroscopic filterable virus. The initial lesion, which has an incubation period of from three to seven days, is small and evanescent. It occurs on the penis in males, and usually within the vagina in females. Ten days to a month following exposure, the draining lymph nodes enlarge. From this point, the manifestations of the disease in the two sexes differ.

In the male (Fig. 1), because of the location of the primary lesion, the superficial inguinal nodes become involved and eventually suppurate; secondary perirectal involvement due to the passage of the virus along the collateral lymphatic vessels may occur. Another explanation for rectal involvement in males is sodomy.

In the female (Fig. 2), however, the location of the primary lesion causes the deep pelvic nodes as well as the nodes surrounding the rectum to become involved; inguinal localization is rare. The perirectal involvement produces fibrotic, progressively obliterating, inflammatory rectal strictures, usually accompanied by perianal elephantiasis and often complicated with multiple fistulae. The diseased rectum produces a sanguinopurulent exudate which discharges from the anus. Slight to moderate constitutional symptoms such as chills, fever, and general malaise may be present. The most serious sequela of venereal lymphogranuloma is rectal stricture, the course of which is a very chronic one.

As in syphilis, unusual sex practices cause the initial lesion to appear in extragenital locations. Accidental infections in surgeons[25] as well as in infants[26] have been reported. The lymphatic involvement in these cases naturally is governed by the location of the primary sore.

Diagnosis—The Frei Test. Frei's discovery of his intracutaneous test for venereal lymphogranuloma has made possible our present understanding of the disease. Although, with our present knowledge, the diagnosis of the typical case of rectal stricture due to venereal lymphogranuloma presents little difficulty, there are unusual cases in which the Frei test is invaluable for purposes of differential diagnosis. Un-

doubted cases of syphilitic rectal stricture are rare indeed; the occasional case which is seen requires careful study. The Frei test is of distinct diagnostic value and essential in these and all other cases in which venereal lymphogranuloma is suspected.

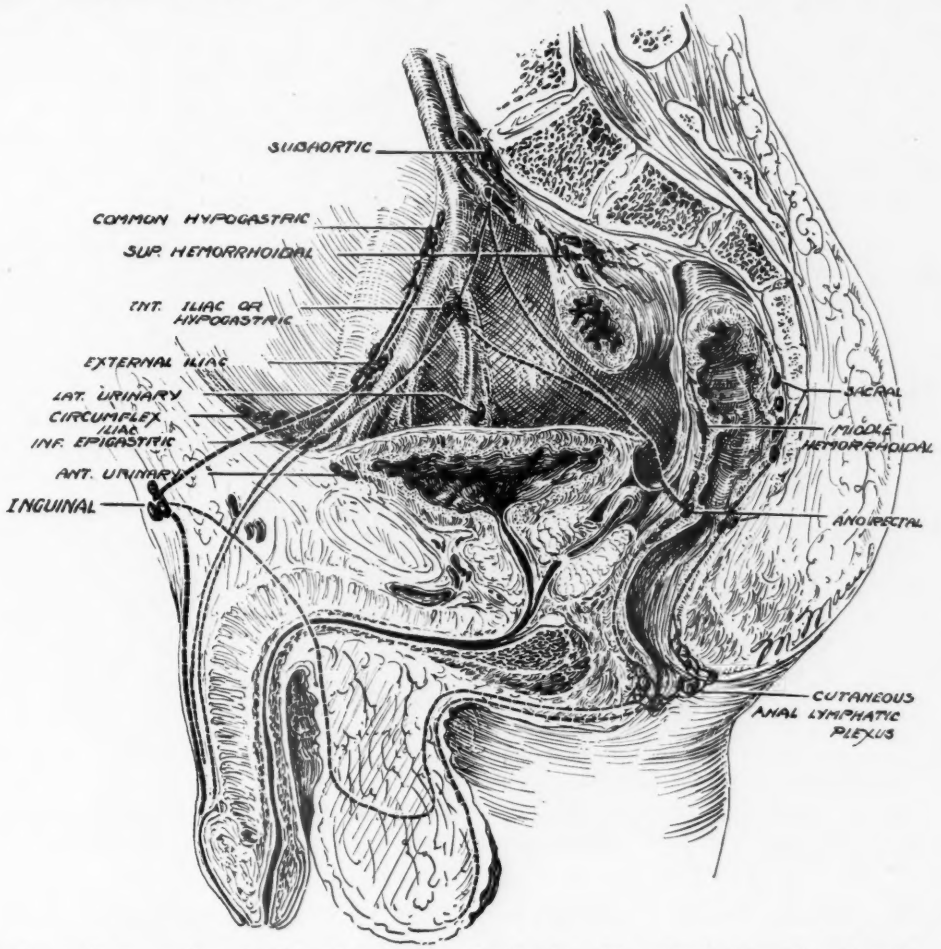

Fig. 1. Cross section of male pelvis showing distribution of lymphatic channels and nodes. After Barthels and Biberstein. (From Lee, H., and Staley, R. W., in *Ann. Surg.*, 100: 490, 1934.)

The test consists of the intradermal injection of 0.1 c.c. of antigen prepared from an uncomplicated, fluctuating node in a known case of venereal lymphogranuloma in which there has not been chancroidal, syphilitic, gonorrheal, or, as Frei insisted, tuberculous infection. The reaction should be read after forty-eight to seventy-two hours at which time it reaches its maximum. A positive test is characterized by the production of an infiltrated inflammatory dome-shaped area from 0.5 to 1 cm. in diameter. The reaction is palpable as well as visible. Of course, control tests are essential. This cutaneous sensitivity lasts for many years, and possibly throughout life.

Preparation of Frei Antigen. According to Frei, the antigen is prepared as follows: Under aseptic precautions, pus from a soft, uncomplicated node (as mentioned above) is aspirated into a syringe. This is diluted and mixed with five to six parts physiologic saline in a sterile tube and immediately put up in 0.5 to 1 c.c. doses in Jena hard-glass ampules. Each ampule is then heated to 60°c. for two hours over a water bath, and the following day at 60°c. for one hour. Before being used, the antigen should be tested for sterility on aerobic and anaerobic media in room and incubator temperature. Tests should then be performed on known

positive cases and normal controls. The potency of the antigen should be tested every three months.

Although the intracutaneous[29,31] and intravenous[32] injections of sterilized Frei antigen offer promise in cases of inguinal

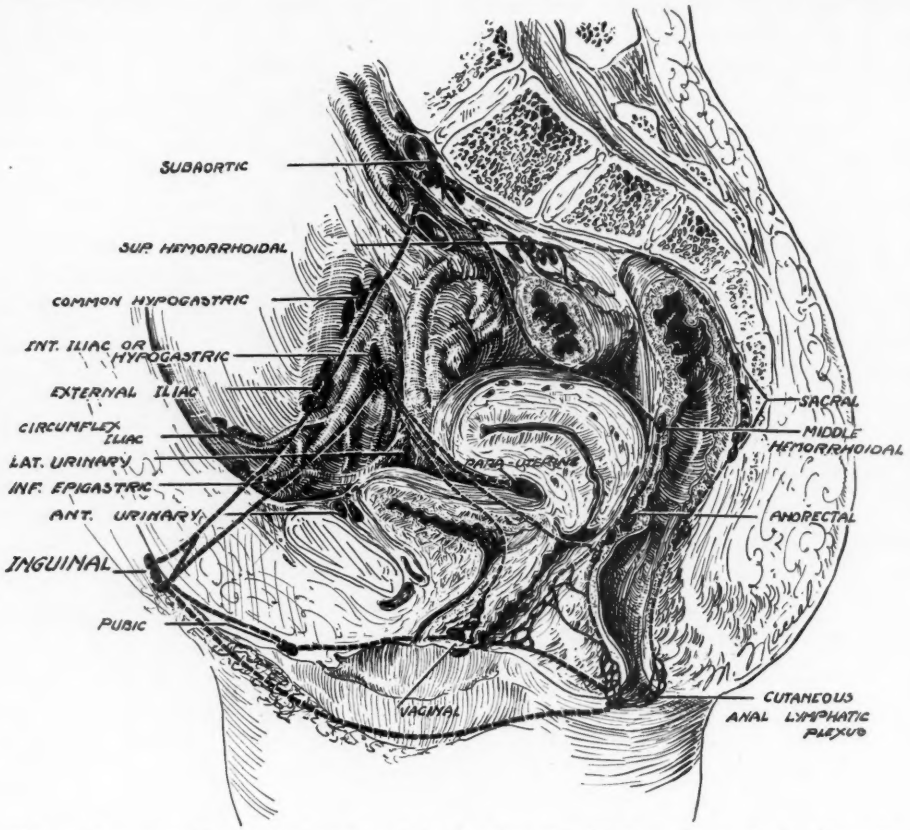

FIG. 2. Cross section of the female pelvis showing distribution of lymphatic channels and nodes. After Barthels and Biberstein. (From Lee, H., and Staley, R. W., in *Ann. Surg.*, 100: 490, 1934.)

Treatment. The remedies suggested for the treatment of this condition are many, but reports of their efficacy are conflicting. No specific has been reported. No routine measure can be recommended. The form and stage of the disease must be considered in selecting therapy. In the absence of suppuration, radiotherapy combined with progressive doses of aqueous solution of iodine associated with sodium thiosulfate is advocated.[27] The favorable results following intravenous injections of tartar emetic[28] and fuadin[29] have not been entirely confirmed. Thomas and McCarthy[30] reported good results in a single case by the use of an autogenous bouillon filtrate applied topically. Hot air and ultraviolet rays as well as injections of patient's own blood and sterile milk have been used.

adenitis, relief from obstruction due to rectal stricture is best obtained by colostomy. For the suppurative lesions, drainage is indicated. Yeomans,[33] in 1933, reported the successful perineal excision of a rectum for stricture which was probably of the venereal lymphogranulomatous variety.

Resection by the abdominoperineal route carries with it a high mortality. Most patients can be made comfortable at least for a time by local surgical measures such as dilatation, posterior proctotomy, internal proctotomy and the excision of elephantiasis and fistulae. We believe, however, that eventually these patients with rectal stricture due to venereal lymphogranuloma must submit to colostomy.

TABLE I

SUMMARY OF FOURTEEN CASES

Case	Race	Sex	Age	Clinical Condition*	Duration		Wass.	G. C.	Frei Test	Treatment
					Sore	Rectal Stricture, Yrs.				
C. J.	C	F	34	Stricture grade 4; esthiomene.	Unknown	3	Neg.	Pos.	Pos.	Colostomy.
A. W.	C	F	46	Stricture grade 4; esthiomene; fistulae.	Unknown	21	Neg.	Neg.	Pos.	Colostomy; local operations; dilatations; tartar emetic intravenously.
V. P.	C	F	41	Stricture grade 2; esthiomene.	Unknown	2	Pos.	Neg.	Pos.	Local operation including internal proctotomy; neoarsphenamine and mercury.
C. C.	C	F	36	Stricture grade 4; esthiomene; fistulae.	Unknown	10	Neg.	Neg.	Pos.	Local operations; refused colostomy.
D. C.	C	F	35	Stricture grade 4; esthiomene.	Unknown	12	Neg.	Neg.	Pos.	Refused colostomy.
M. B.	C	F	31	Stricture grade 4; esthiomene; rectovaginal fistula.	Unknown	4	Neg.	Neg.	Pos.	Local operation; refused colostomy.
J. E.	C	F	38	Stricture grade 2; anorectal fistula	Unknown	3	Pos.	Neg.	Pos.	Local operation; neoarsphenamine and mercury.
C. S.	W	F	49	Stricture grade 4; esthiomene.	Unknown	20	Neg.	Neg.	Pos.	Colostomy; posterior proctotomy; tartar emetic intravenously.
A. G.	W	F	43	Stricture grade 4; anorectal fistulae.	Unknown	18	Neg.	Neg.	Pos.	Colostomy; local operation; tartar emetic intravenously.
M. S.	W	F	30	Stricture grade 2; esthiomene.	10 yrs.	6	Neg.	Neg.	Pos.	Local operation; Frei antigen intracutaneously.
W. B.	W	F	34	Stricture grade 2; multiple abscesses and fistulae.	Unknown	4	Neg.	Neg.	Pos.	Local multiple operations.
K. W.	W	M	36	Stricture grade 2; fistulae.	17 yrs.	2	Neg.	Neg.	Pos.	Local operation.
A. T.	W	M	31	Stricture grade 3; heroin addict 14 yrs.	14 yrs.	4	Pos.	Neg.	Pos.	Posterior and internal proctotomy.
N. T.	C	M	39	Stricture grade 1; penile, scrotal, and anal elephantiasis.	Unknown	7	Neg.	Neg.	Pos.	No surgery. Tartar emetic intravenously.

* Gradation of rectal strictures:
Grade 1: Admitting 1 inch proctoscope with ease.
Grade 2: Admitting ¾ inch proctoscope, but not 1 inch proctoscope.
Grade 3: Admitting ⅜ inch proctoscope with ease, but not ¾ inch proctoscope.
Grade 4: Impossible to introduce, or introduction with difficulty of ⅜ inch proctoscope.

ANALYSIS OF FOURTEEN CASES

We have encountered eighteen cylindrical, inflammatory strictures of the rectum in the last four years. All were subjected to the Frei test. Fourteen of these patients gave strongly positive Frei reactions with different antigens, while repeated tests in the remaining four were negative. Only the fourteen with positive Frei tests will be considered in this summary. Nine of these patients were discovered in the Rectal Clinic of the Brooklyn Hospital, while the remaining five were seen in private practice.

TABLE II

Race	Females	Males	Totals
Colored	7	1	8
White	4	2	6
Totals	11	3	14

Of this group, eleven were females and three were males. The race distribution in the females was seven colored to four white, and in the males one colored to two white. Our finding of such a high incidence of this disease among white people is contrary to the published reports of most writers in the United States. We believe that this seeming discrepancy can be explained by the fact that the percentage of colored applicants to our clinic is relatively low. They are more apt to attend the municipal institutions of our community. We fully realize that no conclusions can be formulated from such a small group of cases as ours, but the findings do emphasize that the disease occurs fairly frequently in the white race.

There were two positive Wassermann reactions among the females, and those occurred in colored women, while there was only one positive Wassermann among the males, and that occurred in a white man. There was no history of chancroid in any of our cases. One of the colored women had a positive vaginal smear for gonorrhea; her rectal smears, however, were negative. No evidence of pulmonary tuberculosis was found in any of our cases, either clinically or on *x*-ray. Rectal smears proved negative for the Entamoeba histolytica.

It has been stated[34, 35] that these patients are from the lower walks of life, but five of our cases definitely did not belong in that category. In fact two of our patients were rather cultured individuals.

Our youngest patient was 30 years of age, and the oldest 49. The average age was about 37. From this it can be deduced that the rectal manifestations in our cases occurred years after the beginning of sexual activity. It is practically impossible to determine with accuracy how soon after the primary infection the rectum becomes involved. These cases are seen only after symptoms such as bleeding, pain, discharge, and obstipation due to partial rectal obstruction occur. Disease in and around the rectum is probably present for years before there are any subjective symptoms.

A definite history of a primary lesion was not elicited in any of our cases. In three cases, however, a history of a small erosion, which we presumed to be due to venereal lymphogranuloma, was obtained. In a male who admitted sodomy, the initial lesion probably occurred in the rectum.

Our patients, according to their histories, harbored these strictures from two to twenty-one years. The average duration was a little over eight years. Generally, as would be expected, the longer the duration of the stricture the more marked and complicated it would be.

Biopsies revealed a remarkable uniformity in the microscopic findings. Sections showed fibrotic tissue containing numerous focal collections of lymphocytes, plasma cells, and other leucocytes, with groups of epithelioid cells in variable numbers. Relatively few foreign body giant cells are present. Different pathologists viewing our sections have called the process infectious granuloma. It was difficult to distinguish the microscopic findings from tuberculosis, syphilis, or other infectious conditions. A knowledge of the clinical findings and a

positive Frei test are supportive in arriving at the histologic diagnosis. The greatest value of biopsy is that malignancy, with which these strictures may be confused clinically, can be excluded.

In the treatment of these cases we have employed the various chemotherapeutic, biologic, and physical measures which have been recommended. We have found them unsatisfactory. Such treatment cannot be expected to alter the effects of fibrosis and cicatrization such as is found in rectal stricture. Surgery is indicated. Colostomy or local surgical measures, or a combination of both, have given our patients symptomatic relief, but the rectal lesions do not show much regression. In cases with imminent obstruction, we have performed colostomies followed by local surgical procedures to establish drainage and reduce the deformity of esthiomene. In cases in which there has not been danger of complete obstruction, we have used such palliative measures as posterior or internal proctotomy in order to postpone the necessity for an artificial anus. Repeated dilatations, in patients with or without colostomy, have not proved of any value. As stated above, we feel that all these cases must eventually submit to colostomy. We have not performed any abdominoperineal resections for these strictures, but it would seem that such operations are feasible.

SUMMARY

Venereal lymphogranuloma, often termed lymphogranuloma inguinale, lymphopathia venereum, etc., is a venereal disease probably caused by a filterable virus. The universal adoption of the term venereal lymphogranuloma would avoid confusion, designate its venereal origin, it would give some conception of the nature of the condition, and embrace the multiple localizations and aspects of the disease.

A history of an initial lesion is difficult to elicit because of the evanescent nature of the primary sore. It occurs on the penis in males and usually within the vagina in females. The manifestations of the disease in the two sexes differ because of the different anatomic locations of the initial lesions. In the male, the superficial inguinal lymph nodes become involved; in the female, however, the deep pelvic nodes as well as the nodes surrounding the rectum become involved. Secondary perirectal involvement may occur in males due to the passage of the virus along collateral lymphatic vessels. In our cases, rectal involvement occurred about four times as frequently in females than in males.

The most serious sequela of the disease is rectal stricture which is the result of perirectal involvement producing obliterating, inflammatory narrowing of the rectal lumen.

This disease probably occurs with equal frequency in temperate and tropical climates. It occurs more frequently in the white race than has been suspected. Just as in syphilis, this disease occurs in people in all walks of life.

The course of the stricture is very chronic; the average duration in our cases was eight years.

Although there is a remarkable uniformity in the microscopic findings, a positive diagnosis cannot be made from biopsy alone.

The Frei test is specific for venereal lymphogranuloma. All rectal strictures require this test.

No specific for the treatment of rectal strictures due to venereal lymphogranuloma has been reported. We believe that these patients must eventually submit to colostomy.

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PERFORATION OF THE GALL-BLADDER

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PERFORATION of the gall-bladder is too frequently considered one of the rare complications in gall-bladder surgery, probably because perforation is usually reported in conjunction with cholecystitis in its chronic state. When one considers the acute gall-bladder and the frequency with which it ruptures, one cannot regard perforation as a rarity. The commonly quoted statistics of perforation show that it occurs in from 1 to 2 per cent of chronic cholecystitis. In 6,800 cases reported by five authors (quoted by Eliason & McLaughlin[1]) there were ninety-six perforations (0.9 to 2.5 per cent). Recently Sanders[2] reported an incidence of 5.2 per cent in a series of 886 cases.

We are reporting a group of seventeen cases in which rupture occurred, in a series of 775 gall-bladder operations done from June, 1925 to January, 1934, the percentage in this series being 2. However, 170 of the total number were acute cases, which brings the incidence to 10 per cent. This is not so high as reported by Heuer,[3] whose figure of 26 per cent is greater than that occurring in Zinninger's[4] series (20.5 per cent) and in Smith's[5] series (22.4 per cent). Judd and Phillips[6] report an incidence of gangrene and perforation in acute cholecystitis of 13.4 per cent, and qualify their findings, stating that because of their location they did not see so many acute cases as surgeons in larger communities. Our incidence of acute gall-bladders presents a figure of 22 per cent, which is more in keeping with that of perforation as reported by the previously mentioned surgeons; but our percentage of perforations we think is still of sufficient size as to make one give much thought to the acute gall-bladder. Seven of the cases occurred in males, ten were in females. The average age was 52; the youngest patient was 23 years of age and the oldest 75. The mean age for the entire group of acute cases without perforation was 46.

Signs and Symptoms. Pain was present in all cases. Practically all had a history of preëxisting chronic cholecystitis. The onset of the present illness occurred usually two or more days before the patient was seen by us. Jaundice was present in but six cases. Twelve had vomited. Chills were noted in but two cases. The average temperature on admittance was 99°F., the highest being 102 degrees, and there were eight cases with normal temperature; temperatures were the same for both the acute and perforated gall-bladders. There was localized tenderness over the gall-bladder in all but two cases, and in these there was generalized tenderness.

The average white count was 14,000, the lowest being 4,500 and the highest 25,000. The mean white count for the total series of acute cases was 11,000, as compared with 14,000 for those with perforated gall-bladders.

Thirteen patients had stones and four gall-bladders perforated in the absence of stones; in only one case were there stones in the common duct. Perforation of the gall-bladder into the free abdominal cavity of a patient in the third trimester of pregnancy has been reported by one of us.[7] Diabetes complicated but one case in this series.

Preparation of Patient. Many times the patient was in such poor general condition that operation was withheld for a period of eight to ten hours, during which time the patient was given large amounts of glucose, saline, and in some cases blood transfusion. It is our belief that in acute gall-bladders,

the sooner surgery is done the better the end result, and this certainly is even more true when perforation of the gall-bladder has occurred. Many times the diagnosis of acute gall-bladder disease is difficult to make, but whenever a patient presents himself with a history of chronic gall-bladder disease, with severe pain in the right upper quadrant, low fever, elevated white count, and with a tender mass or rigidity in the right upper quadrant, surgery should be done promptly. We find it very difficult to diagnose perforation in advance of operation, and consider it even more difficult to predict which gall-bladder will go on to perforation and which will become quiescent, for in our small series we have perforations of the gall-bladder with normal temperatures and normal white counts, and therefore feel the best time to operate on the acute gall-bladder is before it has become either gangrenous or perforated.

Type of Operation. Whether removal of the gall-bladder or simple drainage is to be done depends first, on the patient's general condition and second, on the pathology present. In the aged individual whose general condition is poor, cholecystostomy under local anaesthesia with drainage of the abdomen, is sometimes all the surgery permissible. Occasionally when gangrene is so extensive as to make cholecystectomy extremely difficult, once again the easier of the two operations is done. In this series, eleven gall-bladders were removed and six were drained. Of this latter group, two gall-bladders have since been removed.

Postoperative Condition of Patient. The convalescence, while quite stormy the first two or three days, was frequently not so severe as anticipated. The highest mean temperature postoperatively was 101 degrees. We feel that the patient who has had a suppurative and perforated gall-bladder removed will have a smoother convalescence than one whose gall-bladder was merely drained. The high fever described in the past as liver shock was not seen in any of these cases, which may be explained either by the administration of large amounts of glucose to prevent glycogen depletion of the liver, or by taking particular care that the right hepatic artery is not ligated. Eleven of the seventeen patients had distention which might be described as moderate. There was drainage from the abdominal wound in the average case for twenty-two days, all wounds being considered infected.

Complications. In five cases there were definite chest complications, only two of which were true pneumonia. Diathermy was given with apparent success. Since this series, postoperative inflation of the chest is done routinely and we believe has reduced our incidence of complications of this type. Two subphrenic abscesses occurred postoperatively and were drained; one of these patients died fifty-five days following the original operation and was our only mortality. The diabetic, whose blood sugar at one time was 222 mg., made an uneventful convalescence. The draining infected wounds prolonged the patients' stay in the institution and were the ultimate cause of five incisional herniae. The case of perforated gall-bladder complicating the last stage of pregnancy went on to a very satisfactory termination for both the mother and child.

Site of Perforation. In our experience, the majority of gall-bladders perforate at the so-called "gooseneck," but perforation may readily occur at the fundus, also. The absence of stones does not preclude the possibility of perforation, as in four cases this occurred when no stones were present. Whether or not there is free perforation into the abdominal cavity has much to do with the mortality in perforation of the gall-bladder, according to the literature as reported by Mitchell[8] and Alexander.[9] The figure fifty is the most frequently quoted mortality incidence when there is contamination of the abdominal cavity by bile from a perforated gall-bladder. In our series, perforation into the free abdominal

cavity occurred in six cases without a death. We like to believe that the early institution of operation was a big factor in this achievement. Eleven were walled off by some adjacent organ or structure so as to produce extracholecystic abscess. Two of these eventually went on to subphrenic abscess formation with one death. In none of these cases was there gross hemorrhage into the abdomen.

Mortality. Of the seventeen cases of perforated gall-bladder, there was one death, which makes a percentage of 5.8.

SUMMARY

1. Seventeen cases of perforation of the gall-bladder are presented, with but one mortality.

2. Four cases of perforation of the gall-bladder in the absence of stones were encountered, this being distinctly in disagreement with the early teaching concerning perforation of the gall-bladder.

3. Six cases of acute free perforation of the gall-bladder without a death are reported.

CONCLUSION

A plea is made for earlier operation in acute gall-bladder disease, so that the organ can be removed rather than drained; and complications such as extracholecystic abscess, liver abscess, and fistulae between the gall-bladder and neighboring viscera will not be met, as these are the complications which do much to increase morbidity and mortality in perforation of the gall-bladder.

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THE MORTALITY AND MORBIDITY OF ACUTE APPENDICITIS

A STUDY OF 150 CASES

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IN the four or five decades that have passed since the treatment of acute appendicitis was placed on a surgical basis, many statistics have been given in the literature on the operative mortality of this disease. Mortality rates varying from 3 to 10 per cent are reported[1] from studies of large series. Most of these studies were made on the results obtained on combined ward and private services and probably do not present a true picture of the surgical treatment of appendicitis at its best. More recently a report by Herrick[2] and another by Bliss and Heaton[1] show much lower mortality figures. In the latter series, a study of appendicitis in the army service, most of the cases were seen and operated on early and for that reason perhaps the excellent results were obtained. However, it is a noteworthy fact that in these and other[3,4,5] series with low mortality the great majority of the patients were operated on through a McBurney incision or a modification of it. Reid[6] says that the operative mortality of appendicitis has dropped over 50 per cent in the Cincinnati General Hospital since the McBurney replaced the right rectus incision. Furthermore, these observers follow a certain régime with which it is difficult to disagree.

Every surgeon must have a sort of surgical creed in dealing with appendicitis. It is only through following the tenets of his faith that he may hope to achieve the best results.

This study is based on a review of 150 consecutive private cases treated by operation in one hospital. Every case presented a picture of acute inflammation to the pathologist.

Before reviewing the statistics, one must take into account some of the things which may have a bearing on the results. There are, first of all, factors over which the surgeon has no control. There can be no question that the time elapsing between onset and operation is very important. The taking of cathartics and, possibly, of food aggravates the condition. The age of the individual and his resistance to infection play an important rôle. Finally, the type of infecting organism certainly has to be considered.

Then there is the old controversy of the delayed versus the immediate operation. Without entering into a discussion of the merits of one or the other, we take the side of those who operate as soon as possible or just as soon as the very sick patient is in condition. Nothing except impending collapse should delay removal of the offender. If the removal is done properly there will be very little spread of the peritonitis from the operative procedure. Those patients who come into the hospital with peritonitis, dehydration and signs of circulatory failure should be treated with intravenous solution of 5 per cent glucose in saline plus blood transfusion, until they show improvement.

In our series, the anesthetic used was a combination of regional block with 1 per cent novocaine and adrenalin and inhalation of nitrous oxide, ethylene, or, more recently, cyclopropane.

Except when the diagnosis was in some doubt or when a mass was located too far medially, a transverse muscle-splitting incision was employed. The site of this incision varied a little, depending on the location of a mass but usually it was placed

at the level of the anterior superior iliac spine. Every effort was made to enter an abscess without opening into the general peritoneal cavity.

All fluid was cultured. As soon as the peritoneal cavity was entered, fluid, if present, was aspirated. The cecum was located and the longitudinal bands followed to the base of the appendix. The tip was not delivered unless it came up easily. Quite frequently the appendix was removed in retrograde fashion by ligating its base and severing with a carbolized knife, after clamping securely to avoid spilling its contents. The mesoappendix then was clamped and cut a bit at a time, thus freeing the appendix with a minimum of manipulation. The utmost gentleness was employed to deliver the organ intact. A small light-equipped ribbon retractor sometimes was very useful, but generally retractors and gauze packs were avoided. When the omentum was attached, it was ligated, cut away from the main portion, and left adherent in one piece with the appendix. The stump was inverted with an N-suture of fine silk if there was no inflammatory thickening of the cecum and no abscess or peritonitis. The appendix was removed in every case except one.

All patients with abscess or peritonitis were drained. Where there was enough peritoneal reaction to produce seropurulent fluid with no perforation, closure was done without drainage. The position of the drains was determined to some extent by the findings, but usually one was placed lateral to the cecum, the other toward the pelvis. Large cigarette type drains were used.

In the ordinary case the peritoneum was closed with a plain No. 0 catgut continuous suture. Interrupted chromic catgut No. 0 sutures approximated muscle and fascia and the skin was closed with Bartlett clips. When drainage was employed, one or two dermal stay sutures sufficed to hold the entire abdominal wall down to the peritoneum, the drains coming out of the wound at its lateral aspect. Thus the tissues of the abdominal wall were left free to fight infection and to drain without as much danger of sloughing. Because of the nature of the incision, with its tendency to close rather than open with straining, no fear of eventration was felt.

In the uncomplicated case without drainage, the patient was given water by mouth as soon as nausea had ceased. He received fluid food after twenty-four hours and a soft diet soon afterward. If there was any question of proper ingestion, the giving of food was delayed. Until the patient was able to take fluids by mouth he was given 3,000 c.c. of 5 per cent glucose in saline intravenously every twenty-four hours. For sedation morphine in 1/6 or 1/4 gr. doses was used. On the third day following the day of operation he received an enema if he had not defecated naturally. During the first few days he was made to take a few deep breaths every hour. The Bartlett clips were removed on the fourth day, he was up in chair on the fifth and usually he left the hospital on the seventh or eighth day.

Of much greater importance is the postoperative care of the patient in whom the infection has spread to the peritoneal cavity. It is in this type of case that the mortality and morbidity definitely may be lowered by proper treatment. These patients received absolutely nothing by mouth. If they were distended or were vomiting, the Wangensteen type of suction drainage was instituted. Fluid by mouth was not started until improvement was certain. It is often disastrous to begin the oral administration of fluid and food too early. Morphine was administered hypodermically in 1/6 or 1/4 gr. doses often enough to keep the respirations to sixteen per minute. Every twenty-four hours the adult patient received 3,500 to 4,000 c.c. of fluid intravenously. This fluid was 5 per cent glucose either in saline or in distilled water. More saline was used if there was a large loss of upper intestinal content, less if there was any evidence of edema. The urine output was followed as a good guide to amount of intake. It was kept as near as

possible to 1,500 c.c. daily in adults. The patient was kept in a semi-sitting position. Large, hot, moist dressings were applied to the abdomen at two-hour intervals. These extended from xiphoid to symphysis and covered the entire abdomen. The wound was uncovered entirely with each change. A rectal tube was inserted if there was distention but enemata never were given during the acute stage. Deep breathing at frequent intervals was encouraged and the legs were massaged and exercised twice daily. If the patient was very ill or if his illness was prolonged, he received whole blood transfusions. During the entire convalescence a constant vigilance was kept for complications. Residual abscesses were drained by the most direct route. Most of these were pelvic and were drained through the rectum.

There was a sharp rise in the number of cases in groups from 10 to 30 years of age, with a preponderance in the second decade. (Chart 1.) These findings are in accord with those of others. In contrast, there was a sharp fall in the percentage of cases of ruptured appendices in this age group. (Chart 11.) Whether the very young and the aged are more susceptible to rupture or whether diagnosis is more difficult in these cases has to be considered. Probably both factors enter the picture.

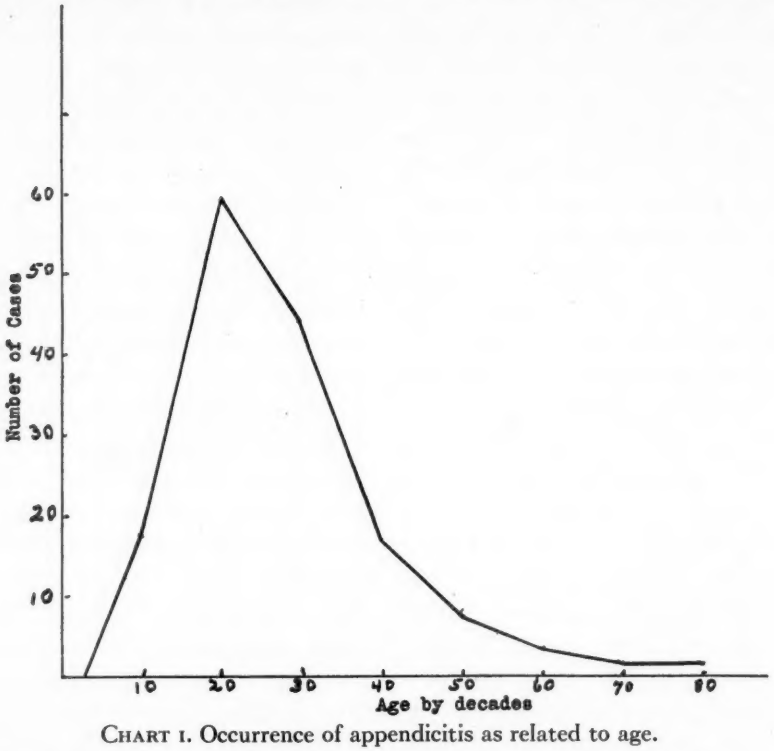

Chart 1. Occurrence of appendicitis as related to age.

The cases were about evenly divided as to sex, seventy-three being male and

Table 1

Sex { 73 male / 77 female

Average Hours Before Operation

Non-ruptured..... 24 (earliest 3 hrs.)
Ruptured..... 104 (earliest 13 hrs.)

History of Previous Attacks

Non-ruptured..... 58—47 per cent
Ruptured..... 11—40 per cent

Average Hospitalization

Non-ruptured..... 8 days
Ruptured..... 19 days

Right Paramesial Incision

11 cases

Ruptured cases { 17 male / 10 female

History of Catharsis

Non-ruptured.... 13 cases—10 per cent
Ruptured..... 12 cases—44 per cent

Average Duration of Operation

Non-ruptured—18 min. { Shortest—8 min. / Longest—40 min.

Ruptured—26½ min. { Shortest—10 min. / Longest—45 min.

Palpable Mass

16 cases

seventy-seven female. (Table 1.) Seventeen of the ruptured appendices were in males and ten in females. In the non-ruptured cases an average of twenty-four hours had elapsed before operation. The earliest was operated three hours after the onset. An average of 104 hours from onset to operation had elapsed in the cases with rupture. One patient showed perforation though only thirteen hours had elapsed and no cathartic had been given.

There was a history of catharsis in twenty-five cases. Among the ruptured appendices, twelve cases, or 44 per cent, followed a cathartic.

Previous attacks had occurred in fifty-eight or 47 per cent of the unruptured cases and in eleven or 40 per cent of those that had perforated.

One of the many advantages of the McBurney type of incision is the ease with which the abdomen may be opened and closed. The operating time, therefore, is cut down, a factor of no little importance in acute cases. The average time for the non-ruptured cases was eighteen minutes. The average of twenty-six and one-half minutes for the ruptured cases shows quite clearly how the operative procedure is affected by the pathology.

Another very definite advantage of the muscle-splitting incision is its ability to stand stress and strain rather early as compared to other types. As a result, patients may be walking on the sixth postoperative day and frequently may leave the hospital on the seventh day. The average hospitalization period for the non-ruptured cases was eight and one-half days. The morbidity of rupture is demonstrated by the prolonged hospitalization. The average in these cases was nineteen days.

There was a palpable mass in sixteen of the cases with rupture. Complete localization was not present, but rather there was some attempt at localization.

A right paramesial incision was employed in eleven cases, either because of some doubt as to diagnosis as mentioned before, or because it offered a more direct approach to a medially placed mass.

Cultures were made of all fluid. There were fourteen patients with fluid but without definite perforation. These were not drained. In one case hemolytic streptococcus was cultured, in one Staphylococcus aureus, and two gave a pure growth of Bacillus coli communis. All of these recovered without complication except for a wound infection with colon bacillus.

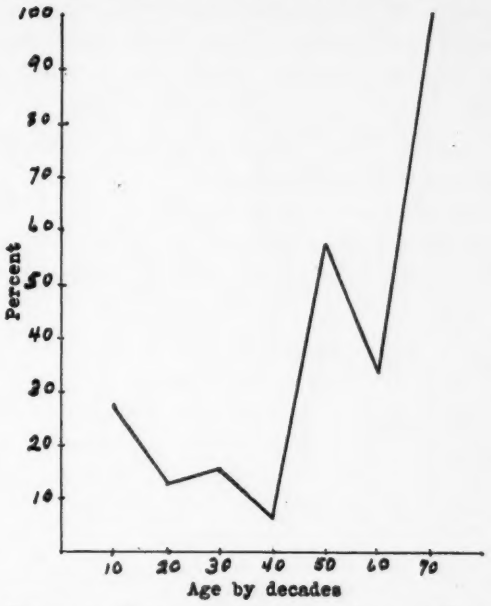

Chart 11. Percentage of ruptured appendices as related to age.

Twenty-five of the ruptured cases gave positive cultures. As far as could be determined from observation of the postoperative course, the type of organism did not seem to affect it except in one patient infected with Bacillus mucosus capsulatus. This very interesting recovery followed six months of hospitalization, during which time three residual abdominal abscesses and a right subphrenic abscess were drained. Each abscess gave a pure growth of Friedlaender's bacillus. At the operation for obstruction which occurred six months after the appendectomy and drainage, a small unsuspected pelvic abscess was found and it, too, gave a pure growth of this organism.

As a general rule, the white blood cell count followed the pathology fairly well,

but in fifteen cases, or 10 per cent of the series, the count was below 10,000. However, a differential count showed more than 80 per cent polymorphonuclear leucocytes in eight of these. Three of these cases showed considerable gangrene of the appendix. In every case with rupture the leucocytes and the polymorphonuclear percentage were increased.

The chart of complications shows how seriously rupture of the appendix affects the outcome. (Table II.) There were only six complications among the unruptured cases. Not even the postoperative pneumonia was serious. On the other hand, all the patients with residual abscesses were quite ill. The patient with multiple complications, mentioned previously as infected with Friedlaender's bacillus, was near death several times during his six month illness. Over half of these cases developed one or more complications before they recovered.

TABLE II
COMPLICATIONS

Non-ruptured Cases	
Wound infection (colon bacillus)	2
Pregnancy (4 and 7 months)	2
Bronchitis	1
Pneumonia (Group IV)	1
Acute otitis media	1
Infarct of lung	1
Postoperative complications	6—4.8 per cent
Mortality of series of 150 cases	0.66 per cent
Mortality of 27 ruptured cases	3.7 per cent
Ruptured Cases	
Residual abscess	8
Fecal fistula	3
Residual abscesses Subdiaphragmatic abscess Femoral phlebitis Intestinal obstruction	1
Postoperative hernia (right rectus incision)	1
Peritonitis 8 days (died)	1
Postoperative complications (including one death)	14, or 52 per cent

One death occurred, in a woman aged 51 who had received chiropractic treatment for a week after the onset of her pain. She had a mass the size of a football in her right abdomen. She was obese and so ill that appendectomy was not attempted. Her abscess was drained and after a period during which she seemed to be recovering, she died of a pulmonary embolus.

The mortality of appendicitis as indicated by this series and others would seem to be the mortality of rupture. So long as the inflammatory process remains confined to the appendix, operative removal is safe and sure, but once the infection spreads to the peritoneum an unfavorable outcome is quite possible.

SUMMARY

A series of 150 consecutive private cases showing the pathology of acute appendicitis is studied for morbidity and mortality.

The method of operation and treatment is given.

Age and sex incidence and other miscellaneous data are charted.

The series shows morbidity in six unruptured and twelve ruptured cases, or 13.3 per cent for the entire series.

The mortality rate is 3.7 per cent for ruptured appendicitis, 0.66 per cent for the series.

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PRINCIPLES OF SURGICAL TECHNIQUE*

WITH PARTICULAR REFERENCE TO THE USE OF SILK

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THE principles of surgical technique here discussed are based to a great extent upon observations and work done at Peter Bent Bringham Hospital, the Harvard Medical School, and in private practice.

Dr. Elliott C. Cutler tells the following story about Dr. Harvey Cushing: When Dr. Cushing had completed his training in surgery at the Massachusetts General Hospital, he received an appointment as assistant to Dr. Halsted at Johns Hopkins. From his training at Massachusetts General, he had come to believe that speed was an important factor, and regarded with considerable pride the fact that he had assisted Dr. J. C. Warren to perform a radical amputation of the breast in some twenty-two minutes. When he went to Johns Hopkins, Dr. Halsted requested him to observe their methods for a few days before beginning to assist. The first morning Dr. Cushing observed an operation, Dr. Halsted was doing an amputation of the breast. When nearly two hours had passed and Dr. Halsted had not completed the operation, Cushing had become rather anxious about the patient's condition. Finally after nearly two and a half hours the operation was completed, and by that time Cushing's anxiety had about reached the limit. Late that afternoon when Dr. Halsted was making rounds he went in to see the patient and found Dr. Cushing there with saline, strychnine, etc., all ready. It was then that Dr. Cushing explained his anxiety concerning the patient and stated he considered her badly shocked. Dr. Halsted told him if she was shocked it could be easily determined by taking her blood pressure. They took the blood pressure and to the amazement of Dr. Cushing it was normal. Then they took up one by one the questions of giving her strychnine and other remedies Dr. Cushing had in the room, and after their discussion, Dr. Cushing was unable to give good reasons why the patient would be benefited by any of them. To Dr. Cushing's surprise and amazement the patient made an uneventful recovery.

Dr. Cushing often tells this story on himself to emphasize that hurry is not a good surgical principle. To be able to operate quickly and at the same time to observe certain well defined principles of surgery such as gentleness and careful hemostasis is fine, but no surgeon should attempt to hurry through an operation in a patient who is a good risk. He cannot hurry and at the same time observe the primary principles of gentleness and careful hemostasis.

There has been voluminous literature during the past two decades or more on the importance of preoperative preparation and postoperative care. I do not desire in any way to detract from the importance of all that has been written; in fact, I think it would be well to reëmphasize and concur in the importance of careful pre- and postoperative measures. But this point I do want to make: if there has been proper preoperative investigation and preparation, and careful technique—including gentleness and hemostasis during operation—then there will be fewer postoperative complications, and the postoperative care will not play so important a part.

* Read before the Seaboard Medical Association, December 8, 1937.

I do not expect all surgeons to agree with some of the principles I follow, particularly in the use of silk. I probably would have disagreed with these same statements one year ago, particularly in reference to speed and the use of silk. I had always attempted to place considerable importance on speed, or perhaps what would be nearer correct would be to say speed to the extent of hurrying, and while I had seen some silk used, I have never been convinced of the logic of its use.

It is not my intention to discuss the technique of different surgical procedures. Dr. Cutler says: "If you are gentle, if you do not infect your patient, if you are careful about hemostasis, and have proper anesthesia, all other things being satisfactory in a good risk patient, you can operate on the patient as long as it is reasonably necessary." After observing his technique and seeing his patients after operation, I am inclined to believe what he says. Speed must never be obtained at the sacrifice of gentleness and careful hemostasis. The surprising thing to me is that Dr. Cutler's patients do not appear to be sick after surgical intervention as my patients used to be, and as those of other physicians frequently are. It is unusual to hear his patients complain to any particular extent; they look well, usually smiling, and the record in reference to temperature, pulse, respiration, etc., seems to bear that out. They do not seem to have the complications and distention I have been used to seeing. The temperature seldom goes above 100.5 F. at any time during their postoperative stay in the hospital. The postoperative appearance of patients, who in the vast majority of cases did not appear really sick, and whose charts indicated their unusually mild postoperative courses (much better than I had been accustomed to see), was the thing that convinced me of the logic of his teachings.

What are the factors in producing such satisfactory postoperative courses?

1. Careful hemostasis.
2. Gentleness.
3. The use of silk.

Of course there are other factors such as asepsis, proper anesthesia, etc. But these are observed by many surgeons who nevertheless are not able to obtain the mild postoperative course that is seen where gentleness and careful hemostasis are stressed, and where silk is used. Gentleness and hemostasis employed in the use of catgut are far different from the same connections when silk is used. The full significance of this is not appreciated until silk is actually employed. With silk a gentleness far beyond what one is accustomed to employ is necessary, because of the delicate nature of the fine silk used. As this gentleness improves, one naturally develops greater consciousness for better hemostasis. Gentleness and careful hemostasis go hand in hand with the use of silk.

I saw a bilateral herniorraphy performed, silk being used on one side and catgut on the other, with silk used in the skin on both sides. After three days the dressings were removed and those in the class who were not present when the operations were performed were asked to examine the wounds to see if they could determine on which side catgut had been used. All five men were able to state definitely on which side each had been used. On the catgut side the width of the area of induration and tenderness along the incision was about twice as great as on the silk side.

Dr. Shambaugh has reported on postoperative wound complications in a controlled series of 2,360 inguinal hernorraphies performed from 1914 to 1934, comparing silk and catgut. When catgut was employed, No. 1 or 2 chromic was used in the fascia, and No. 00 plain for the hemostatic ties. When silk was employed No. 4 black twisted silk was usually used, although occasionally No. 9 black twisted silk was used in the fascia.

In the 2,360 operations there were 108 infections, an incidence of 4.57 per cent. There were sixty-one minor wound complications (hematomas or serum), 2.59 per

cent. There were forty-seven suppurative infections, an incidence of 1.99 per cent.

During the past fourteen years there has been a more extensive use of silk and at the same time an almost continuous decrease in the number of infections, so that out of a total of 511 operations during the past five years there were only seven infections (five suppurative infections and two minor wound complications) an incidence of 1.27 per cent. Everything else being equal, it was found that wound infections occurred 30 per cent more frequently in patients 50 years and older than in patients under 50 years of age. (Table II.) The use of local anesthesia was not found to be a factor in the development of wound infection (Table III), but the incidence of wound infection in the catgut series was twice that of the silk series. In minor wound complications the superiority of silk was still greater, as these minor infections occurred eight times more

TABLE I

INCIDENCE OF FAULTY WOUND HEALING IN CLEAN INGUINAL HERNIORRHAPHIES*

Author	Number of Cases	Wound Infections		Total Wound Complications		Method of Skin Preparation
		No.	Per Cent	No.	Per Cent	
Coley	500	64	12.8	...	....	Iodine
Beckman	308	17	5.5			Not stated
Meleney	502	21	4.1	50	9.9	Ether, soap, iodine
Morian	1407	40	2.8	73	5.2	Not stated
Eliason	749	13	1.7	78	10.4	Soap and mercurochrome
P.B.B.H. series	2360	47	1.9	109	4.5	Alcohol and bichloride

TABLE II

EFFECT OF AGE UPON THE INCIDENCE OF WOUND INFECTIONS*

Age Group	Number of Operations	Suppurative Infections	
		No.	Per Cent
Under 50 years	1730	31	1.79
50 years and over	630	15	2.38

TABLE III

EFFECT OF LOCAL ANESTHESIA UPON THE INCIDENCE OF WOUND INFECTIONS*

Anesthesia	Silk			Catgut		
	Total Cases	Suppurative Infections		Total Cases	Suppurative Infections	
		No.	Per Cent		No.	Per Cent
Local	557	8	1.43	85	2	2.35
General and spinal	727	10	1.37	878	25	2.84

* After Shambaugh, in *Surg., Gynec. & Obst.*, 64: 765, 1937.

TABLE IV

EFFECT OF SUTURE MATERIAL UPON THE INCIDENCE OF WOUND COMPLICATIONS*

Suture	Number of Operations	Suppurative Infections		Minor Wound Complications		Total Wound Complications	
		No.	Per Cent	No.	Per Cent	No.	Per Cent
Silk	1284	18	1.4	8	0.62	26	2.0
Catgut	963	27	2.8	51	5.29	78	8.1
Silk and catgut	113	2	1.77	2	1.77	4	3.0
Totals	2360	47	1.99	61	2.59	108	4.57

TABLE V

EFFECT OF SUTURE MATERIAL UPON HEALING OF SUPPURATIVE WOUND INFECTIONS*

	Number of Cases	Average Hospital Convalescence, Days	Average Total Convalescence, Days
Silk, slight suppuration	5	17	25
Catgut, slight suppuration	12	19	24
Silk, marked suppuration	13	22	42
Catgut, marked suppuration	14	26	46

frequently in the catgut series. (Tables IV and V.)

I quote Dr. Shambaugh's conclusions,

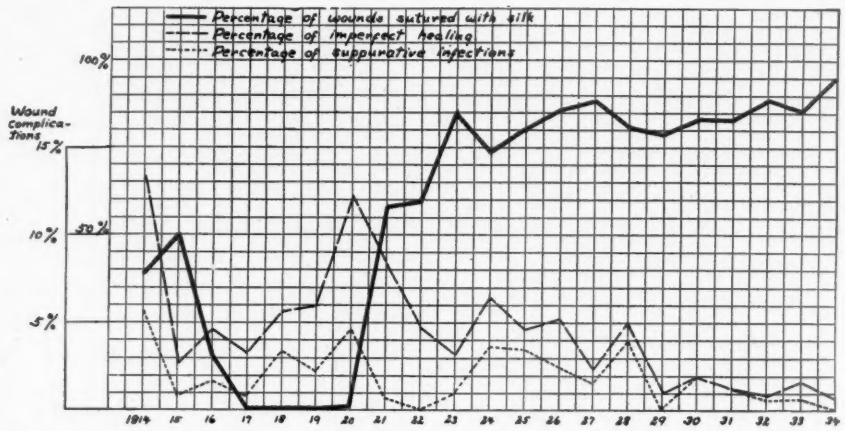
Percentage of wounds sutured with silk
Percentage of imperfect healing
Percentage of suppurative infections
100%
Wound complications
15%
10%
5%
1914 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 32 33 34

FIG. 1. Graph showing relationship between the employment of silk and the annual incidence of wound complications. (From Shambaugh, in *Surg., Gynec. & Obst.*, 64: 766, 1937.)

because they represent the impressions formed by those who have used silk in a large number of cases:

"1. The alcohol and bichloride method of skin preparation compares favorably with other methods as regards the incidence of postoperative wound infection.

"2. Suppuration in clean operative wounds is more likely to occur in elderly patients than in the younger group.

"3. Anesthesia by local injection does not increase the likelihood of wound infection.

"4. In a controlled series the incidence of suppurative wound infections where catgut was employed as a suture material was twice as great as where silk was used.

"5. Where fine silk (No. 4) is used and the principles laid down by Halsted are followed, the presence of silk in suppurating wounds does not, on the average, delay the healing of the wound.

"6. Infected wounds may heal completely and permanently without discharging the silk sutures.

"7. The presence of fine silk in infected wounds may delay healing for periods up to three or four months, but this is exceptional and should not deter one from employing the silk technique in suitable cases."

THE PRINCIPLE OF SILK TECHNIQUE

The following are some of the principles involved in the silk technique:

No. 4 black twisted silk is ordinarily used for most purposes, except in the fascia where a larger size up to No. 9 may be used. The silk should first be dipped in melted bone-wax. The excess wax is wiped off and the silk is autoclaved. It should not be sterilized several times, as it soon loses its strength from repeated sterilizations. A small half-circle No. 2 or 3 French needle is used, except in intestinal work where a straight needle is usually preferable. The sutures are usually cut about 18 inches long. It is best not to use the same suture more than once in a French needle as it may become frayed. A small needle holder must be used, and it is best to clamp the French needle near its middle. The hemostat used for clamping the vessels must be small. One should attempt to clamp only the bleeders; if any tissue is included in the clamp it must be the smallest amount possible. The strangulation of large amounts of tissue must be avoided, but all bleeding must be controlled immediately. The silk suture, the finest that will furnish the necessary strength, must be cut at the knot. Silk sutures are usually interrupted, except in the peritoneum, where they may be continuous. The interrupted skin suture is the only one with which I am familiar

that can be removed without the possibility of contamination of the wound, that is, without drawing some of the exposed with gauze. Silver foil is used to cover the incision and it helps to prevent contamination of the wound.

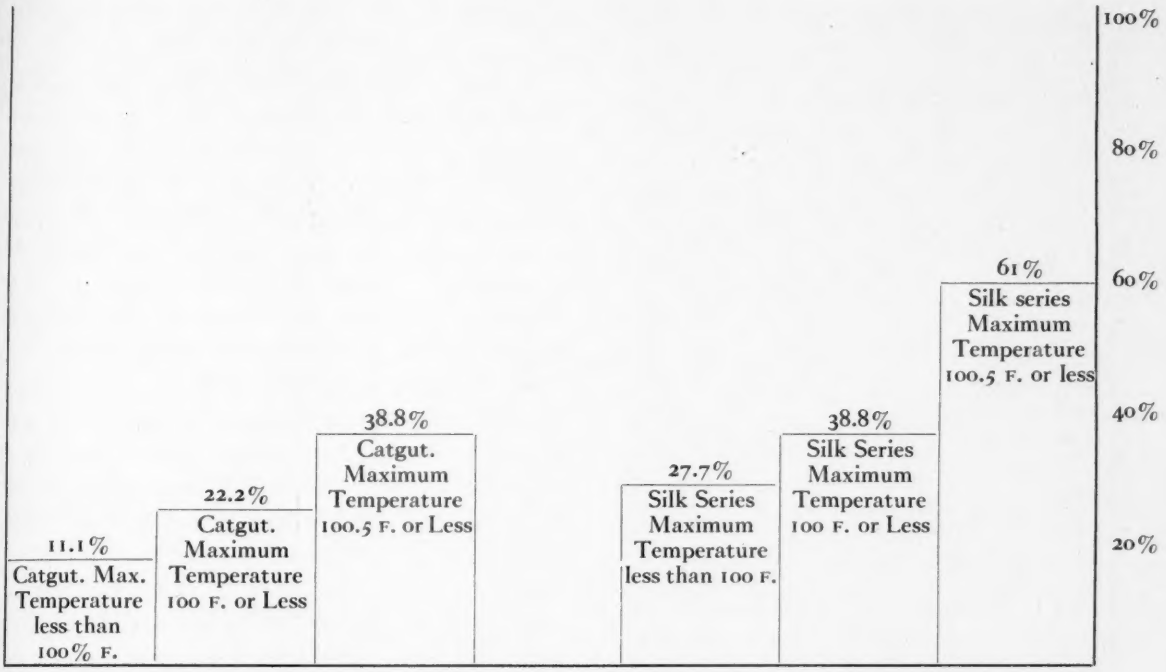

Fig. 2. Graph showing the comparison of temperatures in a series of catgut and silk cases.

part of the suture through the tissues. Skin sutures should be just tight enough to approximate the skin edges; they should not be under tension. A subcutaneous suture is used for the purpose of almost approximating the skin edges, and should be placed so the knot will be underneath. The bite should include only the deeper layer of the skin and should be taken a short distance from the edge of the wound. This subcutaneous suture is very important, because it keeps tension off the skin sutures, helps to produce a narrower scar, and closes dead space. All infections of operative wounds do not occur during the operation; a fairly large percentage occurs afterward from contamination due to carelessness in changing dressings, from carelessness in removing properly placed sutures, and from removing improper sutures. In order to avoid trauma, incisions should be of sufficient length to give adequate exposure. The edges of wound and skin during operation should be protected

PRELIMINARY REPORT

I am well aware of the fact that the small number of cases in this preliminary report cannot be used for the purpose of forming definite conclusions. This report is a series of unselected and consecutive private cases to compare the difference in temperature, etc. when cat-gut and silk were used. In this report there are thirty-six cases, eighteen in each series. In a few cases in the silk series, catgut was used in the peritoneum in order not to delay the surgeon following; catgut was also used in the pelvic part of the operations and around the sites of the drainage wounds. In all cases the peritoneal cavity was opened; there were thirty-three intra-abdominal operations and three inguinal herniorraphies (two herniorraphies in the catgut series, and one in the silk series). Drainage was necessary in six cases: one cholecystectomy, one ruptured appendix, and one tubo-ovarian abscess in each series. There were three cases of acute appendicitis

in the silk series and none in the cat-gut series. There was one case in the silk series where appendectomy was done for chronic

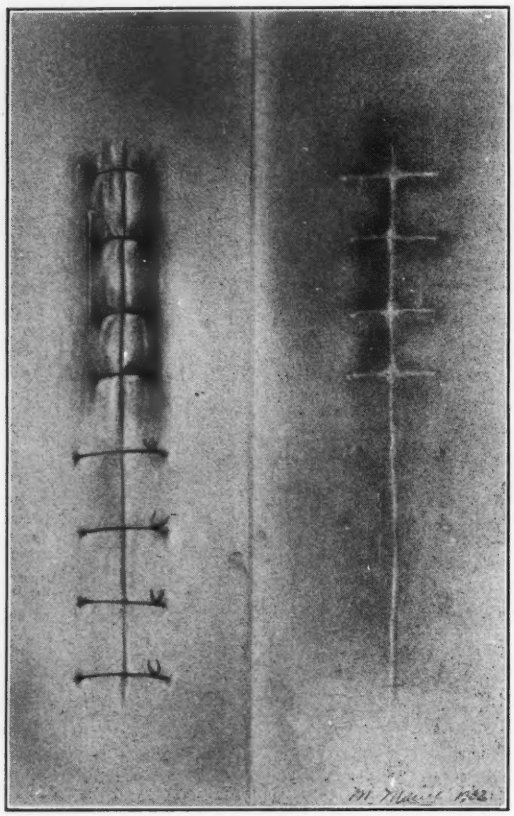

FIG. 3. Difference between the effects of tight and loose skin sutures. The cross scars are the result of anemic necrosis. (From Reid, in *South. M. J.*, 26: 110.)

appendicitis, and there were four such cases in the catgut series. The other operations included appendectomies for subacute appendicitis; supravaginal hysterectomies and various combinations of operations on the internal female organs, which were about equally divided between the two series. As the lowest temperatures are obtained in abdominal surgery in which there is simply an appendectomy for chronic appendicitis, and as there were no cases of acute appendicitis in the catgut series, it seems that if any particular difference existed in the mildness of the conditions, it was in favor of the catgut series.

As indicated by Figure 2, there were definitely lower temperatures in the silk series, but because of the small number of cases no definite conclusions can be reached; however, if this same advantage for silk could be maintained in a large series of cases it would be a most important point in favor of the silk technique.

In the catgut series 11.1 per cent had a maximum temperature of less than 100 F. In the silk series this was true in 27.7 per cent. In the catgut series the maximum temperature was 100 F. or less in 22.2 per cent; in the silk series in 38.8 per cent. A maximum temperature of 100.5 F was observed in 38.8 per cent of the catgut series and 61 per cent of the silk series.

If, when using the silk technique in clean cases, the postoperative temperature goes above 100.5 F., one should look for complications. They will not always be present, but with the silk technique temperatures are usually low, and a rise above 100.5 F. often indicates that something is wrong. The maximum temperature in the patient who had the least elevation of temperature in the catgut series was 99.8 F. The diagnosis was chronic appendicitis. The maximum temperature in the patient who had the least elevation of temperature in the silk series was 99.4 F. The diagnosis was chocolate cyst of the left ovary; cystic right ovary; chronic appendicitis, and partial stenosis of the cervical canal. There were three cases of acute appendicitis in the silk series and the maximum temperature did not reach 100 F. in any case.

No evidence of infection occurred in any of the clean cases in the silk series. In the drainage cases in the silk series there was no infection except around the drain, and the drainage wounds healed promptly with no evidence of infection in any other part of the incisions.

The duration of operations when using the silk technique is usually from twenty-five to fifty minutes longer than when using catgut, and because of the longer duration of the operations it is advisable to use basal anesthetics more often. In this series avertin was used as a basal anesthetic, in doses of 60 or 70 mg. per kilo of body

weight. Avertin was not used when there was a diseased condition of the biliary system or kidneys, or in hypotension.

The coöperation of the interns, anesthetist and operating room nurses is absolutely essential. The patient is the most important person in the operating room during the operation, and everyone connected with the operation should pay particular attention to the various steps so there will be no unnecessary delay.

CONCLUSIONS

From this preliminary report no definite conclusions can be reached in regard to the use of silk; however, it does seem that there is some justification for a favorable impression from the results obtained in the silk series. Certainly the lower the temperatures, the more comfortable the patients and the fewer the complications.

The milder postoperative courses observed when using the silk technique seem to result from a combination of circumstances, of which silk is the most important, as its use commands greater gentleness and more careful hemostasis.

While there were lower temperatures in the silk series, the things which impressed me most were the general well-being of the patients and a milder postoperative course than I have been able to obtain before. There also seemed to be less distention and fewer gas pains.

From the experience of those who have used the silk technique in a considerable number of cases and from my own limited experience, it appears that silk has a definite superiority over catgut in regard to wound complications.

Trauma, hemorrhage, and infection are the big factors in producing postoperative discomfort and complications.

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CASE REPORTS

INGUINAL HERNIA DUE TO DIRECT TRAUMA: TECHNIQUE OF OPERATION

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INGUINAL hernia as a result of direct accidental trauma is rare and the technique of repair must be adjusted to the extent of laceration of the tissues.

Traumatic hernia may be a result of either direct or indirect trauma. It is usually medial, but may be lateral to the inferior epigastric artery or involve both regions. Hernia resulting from direct trauma may be due to laceration of the protecting structures, in an accident, at operation or from intentional self injury. Hernia from indirect trauma may result from an increased abdominal pressure with a predisposing patent peritoneal process within the cord or to a congenital or acquired weakness of the protecting muscles and fascia.

Erdman[1] stated that the enlargement of the external ring which was present in 2.2 per cent of drafted men may be a determining factor in direct hernia. A definite history of accidental indirect trauma was obtained by Fallis[2] in 62.1 per cent of 994 patients with hernia. Gorton[3] believed that the hernia of weakness in the absence of a preformed sac was rare. Moorhead[4] concluded that a hernia cannot develop in the absence of a preformed sac, a congenital predisposition or an acquired anatomic defect, but that trauma can aggravate, accelerate, accentuate, increase or modify it. St. Jacques[5] believed that hernia following a direct blow to the abdomen was rare, but conceded that its sudden development following effort should be considered as distinct from the aggravation of a preëxisting one. The type of hernia is frequently difficult to diagnose until the relation to the inferior epigastric artery or the cord has been determined and is frequently a complicated or pantaloon type of sac.

Bull and Coley,[6] after investigating the alleged relation of injury to hernia in 10,000 cases, considered that only two were due to direct violence. Selby[7] reported a case of direct hernia following a direct injury in which operation disclosed a tear in the external oblique fascia, the internal oblique and the transversalis muscles.

Holland[8] has submitted six characteristic points for the diagnosis of traumatic hernia: immediate descent of the hernia; severe pain in the hernial region; marked prostration; symptoms of such severity that attention is called to it within twenty-four hours; absence of a history of a previously existing hernia; and evidence that the trauma or strain was adequate. Following direct accidental traumatic hernia there is usually visible evidence of trauma, such as superficial abrasions, edema, subcutaneous ecchymosis, hematoma, redness or increased warmth to the area.

REPORT OF CASE

B. P., a male of 22 years, entered the hospital August 20, 1936. He complained of a mass in the left groin which developed immediately upon being struck there by the handlebar of his motorcycle when he ran into a tree and was thrown to the ground.

When he stood up the pain was so severe in his left inguinal region that he had to lie down.

In a short time he was able to get up and he noticed a mass in his left groin. The next day the mass became smaller when he was lying down and could then be pushed back, but it recurred upon standing. The patient was examined by a local physician thirty-four hours after the accident. He was unable to work but could walk about his home. One week after the injury the swelling did not subside upon lying down and he could no longer push it back.

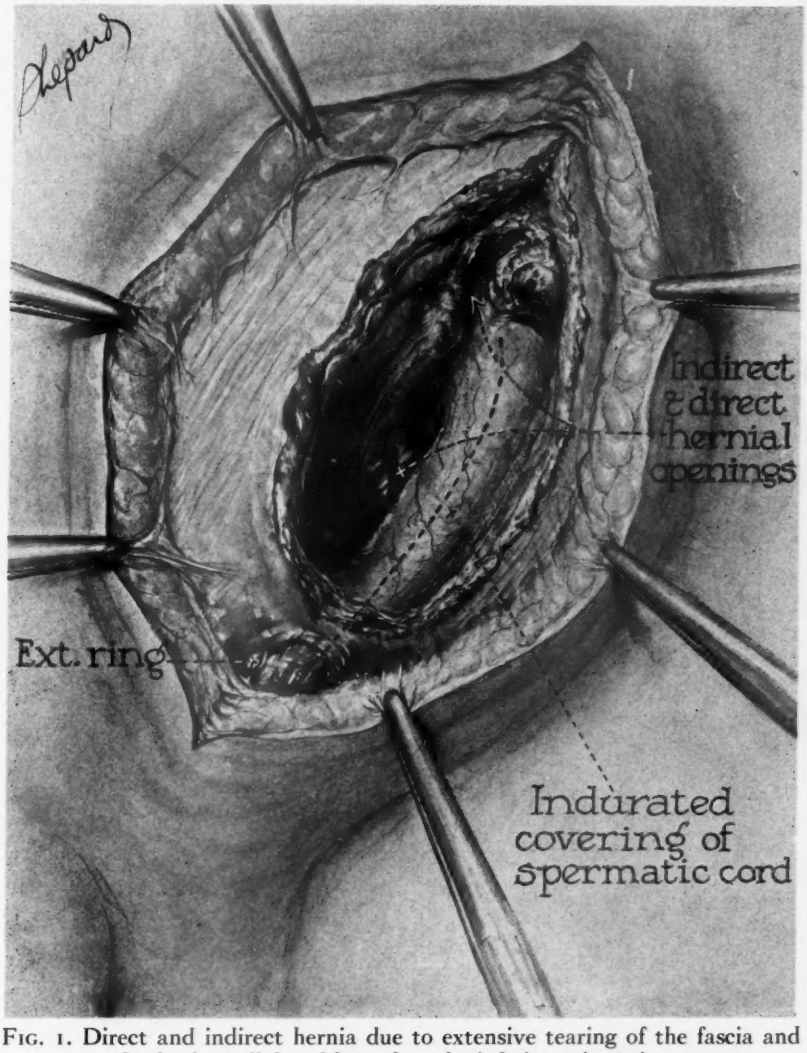

Fig. 1. Direct and indirect hernia due to extensive tearing of the fascia and muscles both medial and lateral to the inferior epigastric artery.

Three months before the accident the patient had been examined by the physician at the factory where he worked and no hernia was discovered.

A sharply outlined mass could be noted on the left side, limited below by Poupart's ligament. It extended medially to the tubercle of the pubis and laterally to within about 1 inch of the anterior superior spine of the ilium. This mass could not be reduced in size by pressure. There was an indurated, slightly discolored area in the skin just lateral to the pubic spine, indicating the site of a recent injury, and below this an area of subcutaneous ecchymosis. The swelling extended 2¾ inches above and 3¾ inches parallel to Poupart's ligament. It was elevated about 1 inch, while the superior and lateral borders were less abrupt than that above Poupart's ligament. The external inguinal ring, which was just medial to the mass, admitted only the tip of the index finger. Both testicles were equal in size with no evidence of injury. Peristalsis was heard distinctly over the swelling and there was definite bulging upon coughing.

At operation on August 24, 1936, a complete tear was found in the external oblique fascia above and extending laterally a short distance beyond the inguinal canal, except for a few fibers forming the external ring. The underlying internal oblique and transversalis muscles and the deep fascia were torn through, producing a defect in Hesselbach's triangle 1 by 2 inches in extent, extending laterally beyond the inferior epigastric artery 1½ inches and surrounded by about 30 c.c. of old blood serum. The torn muscle fibers were indurated and the cord which extended below the hernia was covered with a dense layer of fibrin (Fig. 1.) The torn structures were identified medial and lateral to the inferior epigastric vessels, but due to the induration it was impossible to suture them to Poupart's ligament at its medial end.

The conjoined tendon was sutured with chromic catgut to Cooper's ligament along the ileopectineal line and the internal oblique and transversalis muscles to the outer portion of Poupart's ligament beneath the cord. The defect in the muscles lateral to the inferior epigastric vessels was repaired by suturing the ends of the transversalis and the internal oblique muscles together with mattress sutures. (Fig. 2.) The edges of the torn external oblique fascia were sutured together beneath the cord with imbrication but without constriction by making a short vertical incision in the underlying edge of fascia. The subcutaneous fascia and skin were sutured over the cord. The patient was discharged without complications on the fourteenth day.

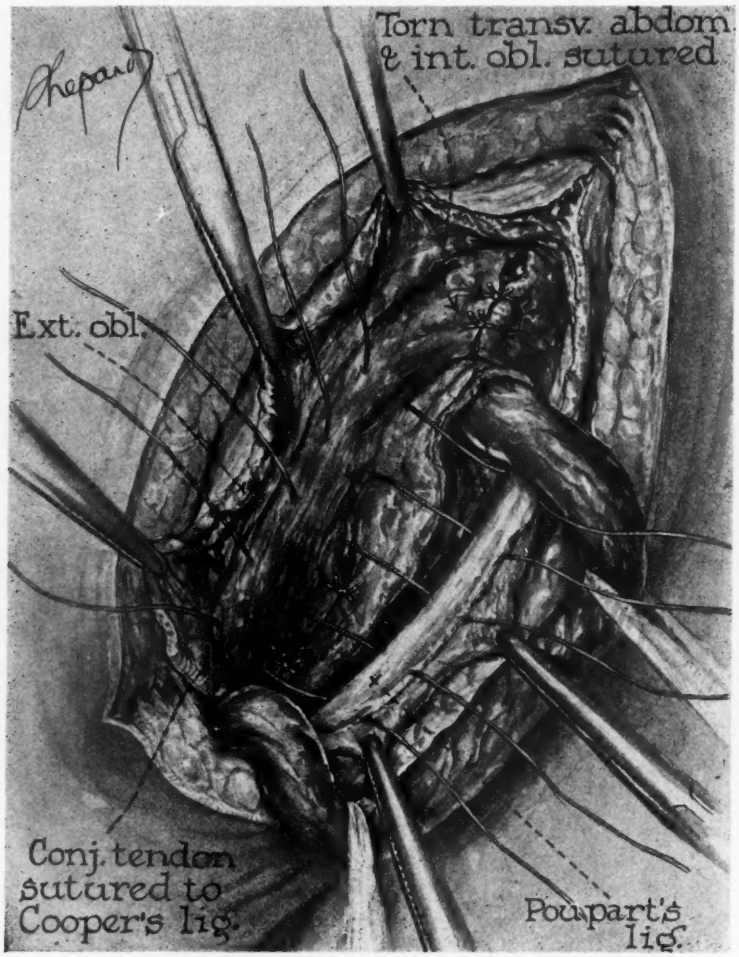

FIG. 2. Conjoined tendon sutured to Cooper's ligament. Muscles and fascia repaired and sutured to Poupart's ligament beneath the cord. Constriction avoided by short vertical incision in underlying fascia at new exit.

Examination six months later showed a firm abdominal wall although the patient had been

doing heavy labor for the preceding four months.

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The outstanding facts concerning the effects of pituitary extract on the metabolism of fat appear to be (1) large doses of anterior pituitary extract bring about a deposition of fat in the liver . . . —and (2) produce ketosis which is probably associated.
From—"The Physiology and Pharmacology of the Pituitary Body," vol. 2, by H. B. Van Dyke (University of Chicago Press).

RESECTION OF CARCINOMA (CARCINOID?) OF THE INFRAPAPILLARY PORTION OF THE DUODENUM INVOLVING THE AMPULLA OF VATER*

REIMPLANTATION OF THE COMMON AND PANCREATIC DUCTS

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JUDGING from the number of cases reported in the literature carcinoma of that portion of the duodenum distal to the ampulla of Vater is quite rare. Lieber, Stewart and Lund have recently reviewed the literature on this subject and of sixty-two cases recorded, only twenty-eight reports were considered sufficiently descriptive to be acceptable as instances of this condition. To this series they added two cases observed among 23,500 autopsy records in Philadelphia. Of twenty-one patients operated upon, thirteen died within fifteen days of the operation of complications directly associated with the surgical intervention. Of the remaining eight, five died within two to three months, two were reported alive three months after operation and in one no postoperative details are given.

Some of the procedures followed were: gastroenterostomy in twelve cases (accompanied by cholecystogastrostomy or duodenostomy in two instances); cholecystostomy alone in one case; and resection with anastomosis in six cases. Of the latter group two were alive after three months and a third died three months after the operation.

Because certain conditions are of such rarity that it falls within the province of most individual surgeons to meet with them perhaps only once or twice over a span of many years, it is felt that individual reports of such conditions are justified. In this manner a composite record of experiences may be compiled. For these reasons the following case is recorded:

R. H., white female of 41, married, was admitted to the University of Chicago Clinics on the medical service of Dr. George F. Dick October 22, 1937. For the previous two years the patient had been aware of increasing fatigability to the point where by 10 A.M. it was almost impossible for her to continue with her domestic work which she had been able to do without difficulty. No palpitation, shortness of breath, edema about the ankles, or tremor was discernible.

Synchronous with the above symptoms there appeared to be an increase in quantity of stool and irregular periods of frequent stools. On one or two days there would be five to seven mushy stools and lower abdominal pains followed by two to four days when there would be only one stool. The character of the stools had also changed; they were very "sour" and foul and varied from clay-colored to dark brown. No gross blood or tarry stools were ever noted. The appetite remained good, but during the two years the patient had lost ten pounds. No history of icterus was given. The family history was irrelevant. Four children were living and well.

Physical examination revealed as well developed, slender, middle aged white female, not acutely ill. Peristaltic waves about the umbilicus were visible, but for the patient's weight (96 pounds) this did not appear necessarily abnormal. To the right and slightly above the umbilicus a vague deep mass about the size of an egg could be palpated sometimes and when it was felt the patient complained of distinct tenderness. No cervical or inguinal

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adenopathy and no icterus were present. The physical findings were otherwise negative.

Blood pressure was 108/70, pulse 78, temperature 98.6°F. The Wassermann and Kahn tests were negative. Red blood cells numbered 4,200,000 and the white 13,000. Hemoglobin was 82 per cent. A differential count showed 73 per cent polys, 21 lymphocytes, 3 monocytes, and 1.2 per cent reticulocytes. Urinalysis was negative. Multiple stool examinations showed light to clay-colored stools very foul, and of sour odor, containing much fat; occult blood was also present. The sugar tolerance curve was normal and the basal metabolic rate minus 10. Free acid was present in the Ewald meal. Blood chemistry showed: 176 mg. per cent cholesterol; 9.7 mg. per cent serum calcium; 4.4 mg. per cent serum phosphate.

Roentgenographic examination revealed a tumor mass approximately 8 by 5 cm. lying within the distended lumen of the second portion of the duodenum. The colon, stomach and esophagus were negative.

The preoperative clinical diagnosis was a neoplasm of the pancreas or duodenum with partial obstruction of the pancreatic ducts.

Operation was done under spinal anesthesia on November 26, 1937. The abdomen was entered through a high midline incision. No free fluid was present. The gall-bladder was markedly distended by bile but its wall appeared normal; no stones were palpable. Exploration was negative except for an oval tumor mass in the lower part of the descending duodenum. The latter was quite mobile. The transverse colon and mesocolon were lifted upward and covered with moist towels. The small intestines were packed downward and to the left in a similar manner. With the tumor bearing segment of duodenum immobilized by the left band a longitudinal incision 8 cm. long was made through the anterior duodenal wall over the tumor which appeared to arise from the posterior wall. When this wound was spread, a lobulated grey tumor mass with several shallow ulcerations upon it was exposed. Palpation revealed it to be quite firm—circumscribed and movable in the duodenal wall. The gross characteristics were those of benign tumor. It was felt at the moment that the neoplasm was essentially submucosal; when pulled upward it appeared to have a thin elongated pedicle of mucosa. There was no evidence of infiltration about the base. Inspection of the duodenum above the tumor failed to reveal the ampulla of Vater. The tumor was pulled upward again and with scissors dissection was "shelled out" by cutting transversely through the pedicle from

Fig. 1. Roentgenogram showing large filling defect in duodenum due to neoplasm in lower second portion. S, stomach. D, duodenal bulb.

left to right. When the last 1 or 2 cm. of the pedicle were cut, a small gush of clear fluid was noted and then a vigorous gush of bile. The latter was sponged and its source revealed as peritoneum closed over this in a separate layer. Palpation of the now relaxed and partially filled gall-bladder again revealed no stones. The abdominal wound was closed in layers.

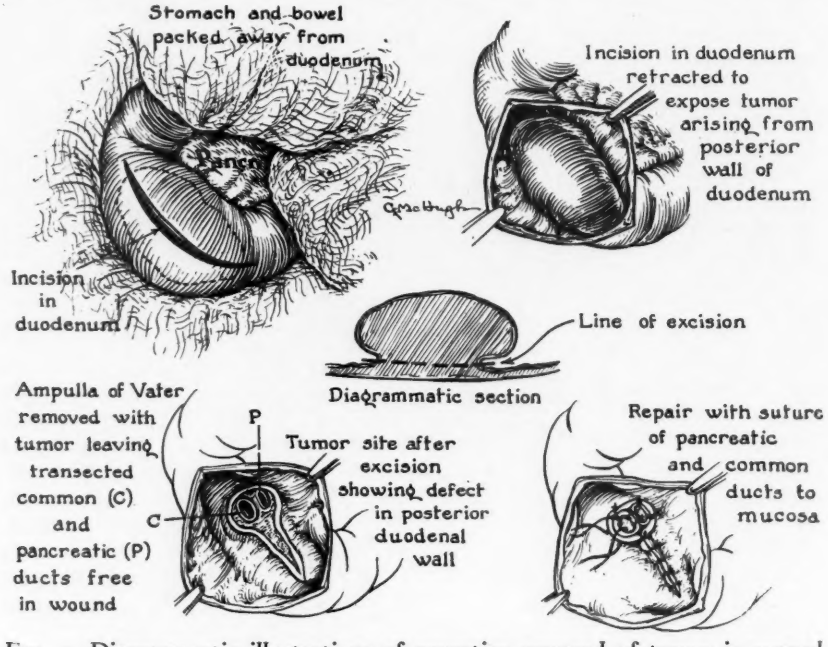
Stomach and bowel packed away from duodenum
Pancreas
Incision in duodenum
Incision in duodenum retracted to expose tumor arising from posterior wall of duodenum
Line of excision
Diagrammatic section
Ampulla of Vater removed with tumor leaving transected common (C) and pancreatic (P) ducts free in wound
P
C
Tumor site after excision showing defect in posterior duodenal wall
Repair with suture of pancreatic and common ducts to mucosa

Fig. 2. Diagrammatic illustrations of operative removal of tumor in second portion of duodenum.

the transected common duct lying free in the right portion of the elongated narrow defect in the posterior duodenal wall resulting from the excision. A small suction tube was inserted into the duct which kept the field dry of bile. Just above the common duct, also lying free in the wound, the thin walled duct of Wirsung was identified. Both ducts were then immobilized by clamping of their edges with mosquito forceps, care being taken to include only a minimum of tissue.

As stated, it was the impression at this time that the muscularis of the posterior duodenal wall had not been completely transected, and because the excised base of the pedicle of the tumor left but a narrow elongated wound no probing through the latter was performed, there being no gross evidence of remaining neoplasm. Using fine "nerve silk" the common and pancreatic ducts were sutured together by two interrupted stitches and in a similar manner sutured to the surrounding mucosa. The remainder of the wound was closed by a single layer of interrupted silk sutures of heavier grade. The linear incision through the anterior duodenal wall was closed by two rows of interrupted silk sutures and the posterior parietal

There was a minimal reaction after operation, the highest temperature being 100.4°F. on the second day.

Following operation there was, of course, little change in the character of the stools. The patient was followed in the Out-Patient Department and by February 18, 1938, had gained 10 pounds and felt generally much improved. She was eating liberally of a general diet. Fatty stools continued. When the patient was last seen in February, 1939 the improvement had been maintained and the only complaint was an excessively fatty stool every three to four weeks.

Histologic Study. A section through the neoplasm, including overlying duodenal mucosa, showed atrophic and degenerative changes in the latter due to pressure from the neoplasm below. The tumor itself was composed of anastomosing broad cords of small, closely packed, polyhedral or cuboidal cells with rather dense eosinophilic cytoplasm and large rounded nucleus rich in finely divided chromatin. In some areas the cells were grouped about a lumen, forming alveolae. Only an occasional mitotic figure was present. Between the masses and cords of neoplastic cells were bands of

smooth muscle—the muscularis of the duodenum. The extent of the latter in the sections made it highly probable that the tumor had infiltrated through the entire duodenal wall

Fig. 3. Gross appearance of excised neoplasm. The metal probe is in the main pancreatic duct and the wooden probe in the common bile duct.

and was not essentially within the submucosa as was thought to be the case at operation.

The impression gained upon study of the routine sections in conjunction with Dr. Eleanor Humphreys of the Department of Pathology was that the tumor resembled more a carcinoid tumor than any other type of epithelial neoplasm and that it was not typical of any of the usual varieties of carcinoma of the bowel. Sections were stained for argentaffine cells by Dr. Humphreys but none was found. Mucicarnine stains revealed no mucus in the cells.

The pathologic diagnosis was carcinoma, but quite possibly carcinoid tumor, of the second portion of the duodenum, with involvement of the ampulla of Vater.

DISCUSSION

1. *The Histologic Character of the Tumor.* An absolute diagnosis of carcinoid could not be made in the absence of argentaffine granules, although, as stated, the hematoxylin and eosin sections strongly suggested this type of neoplasm. The tumor in all probability was a slowly growing malignant one. From the history it had certainly been present for more than two years. It is generally held that carcinoids, especially those not in the appendix, are of low grade malignancy and, if not removed, eventually produce metastases.

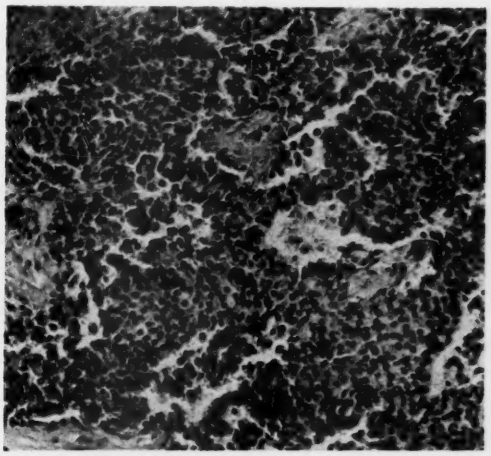

Fig. 4. Photomicrograph (× 60) showing neoplasm to be composed of small closely packed epithelial cells.

2. *The Clinical History.* Carcinomas of the ampulla or of the duodenum immediately adjacent to it usually produce obstructive jaundice which is generally considered a cardinal clinical sign of ampulla tumors. In this case the clinical picture was essentially one of steatorrhea due to slowly increasing obstruction of the main pancreatic duct. There was also, of course, encroachment upon the common bile duct, which in this case appeared in the ampulla to empty separately into the duodenum. No clinical evidence of biliary obstruction was observed. It would appear from the findings and the history that the neoplasm began in the duodenal wall a short distance below the ampulla of Vater and involved the latter during subsequent extension.

3. *The Effects upon the Pancreas.* It has been well established that in experimental animals ligation of the pancreatic duct is followed, among other things, by degeneration of acinar tissue of the pancreas. While in the above case pancreatic tissue was not removed, there was in all probability extensive degeneration of the pancreas. Because of this it is to be expected that steatorrhea would continue for some time, until sufficient acinar tissue has reformed

to supply adequate pancreatic juice for normal digestion.

It would appear that Halsted in 1898 was the first successfully to remove a portion of the duodenum and reimplant the common and pancreatic ducts. The patient lived seven months and died from recurrence of the lesion.

In a review of the recorded instances of radical operations for carcinoma of the periampullary region of the duodenum from 1925 to 1934, Hunt and Budd report four deaths in eighteen cases or a mortality of 22 per cent.[2] A review by Cohen and Colp[1] of cases previously reported in the literature showed a gross mortality of 43 per cent in fifty-eight instances where a variety of procedures was followed.

SUMMARY

An instance of a large, slowly growing carcinoma (carcinoid?) of the second portion of the duodenum secondarily involving the ampulla of Vater is recorded. Transduodenal resection and reimplantation of common and main pancreatic ducts were performed. The patient remained free from evidence of recurrence fifteen months after operation.

The special clinical feature of the case was that steatorrhea due to obstruction of the pancreatic duct was the outstanding sign and no clinical evidence of common duct obstruction was present.

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OSTEOGENIC SARCOMA ARISING IN A DEVITALIZED PART FOLLOWING INJURY

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At the present time the etiology of sarcoma is still surrounded by mystery. The theory of chronic irritation holds weight as a probable factor in many anatomic sites, but, whether the injury of a single trauma is sufficient to induce tissue neoplasia is still a very moot question, and one which frequently baffles the surgeon engaged in traumatic and industrial surgery.

The case herein presented is of clinical and legal interest because the relationship of trauma to tumor was complicated by the fact that the part had suffered devitalization for many years. Although medical testimony was controversial, legally the case was upheld as compensable.

Ewing[1] is of the opinion that chronic irritation is the type of injury that may be a factor in the production of malignant neoplasia.

William B. Coley,[2] in the year 1897, presented a paper before the New York Surgical Society, entitled, "The Influence of Injury upon the Development of Sarcoma," in which he analyzed 170 cases of sarcoma which were personally observed by him, forty-six of which gave a definite history of previous local injury. In 1910, in another publication,[3] he reported 970 cases of sarcoma personally observed, in which there was a history of previous local trauma in 225 cases, or 23 per cent; and 250 cases of carcinoma in which there was a history of injury in eighty-two cases, or 32.8 per cent.

Coley and Higinbotham[4] in a recent article contend that there is a direct relationship between trauma and malignant tumor based upon a hypothetical micro-organism theory. They state: "We may assume that malignant tumors in man are due to a micro-organism that is latent in the circulation, and which gives rise to symptoms of malignancy only after the normal resistance of the cells is broken down, in some instances by local trauma. The micro-organism thus finds a suitable soil in the damaged cell, forming a symbiosis with the cell and causing a proliferation and multiplication resulting in a malignant tumor."

Contrary to this belief, Knox,[5] in 1929, definitely ruled out trauma as an etiologic source of tumors. She placed considerable weight upon the inability of a single trauma to produce tumors experimentally. Lubarsch and Ribbert were both unsuccessful in producing neoplasia, whether by a simple traumatization of tissue or by the injection of traumatized tissue into the animals. Knox reasoned that as a result of a single trauma scars may persist as demonstrable lesions either because of the presence of foreign material implanted in the beginning, or possibly resulting from an infection. Therefore, the important factor seems to be that of chronic irritation rather than acute single trauma. Such a sequence would obtain support in the report of Bloodgood,[6] who found that of forty-eight sarcomas of the skin, the neoplasms developed in a nevus eight times, in a scar twenty times, and in a fibroma nine times.

Lewy[7] studied 23,389 injuries in one year and found only thirty-seven tumors. From this extensive review he believes that trauma is not a primary cause of tumor.

William Seaman Bainbridge[8] writes that it is conceded that surgeons with the full facts assembled before them—clinical his-

tory, signs, symptoms and laboratory reports—are the ones to give the decision in any case as to whether the neoplasm

Fig. 1. Appearance of patient before amputation, showing marked edema and deformity of left arm.

which follows the trauma is the direct result of the injury. Similarly, Kaplan and Rubenfeld,[9] in discussing soft tissue sarcoma, state: "In our opinion, based entirely upon clinical factors, the patient, the tumor, and the traumatic effect must, in every instance be individualized, since the sequence is apparent in some cases and dubious in others."

CASE REPORT

A. K., 25 years old, a white male, presented himself for examination, on April 10, 1934, with a history of having fallen from a wagon on February 5, 1934. He struck the anterior surface of the lower third of his left arm, at which site there was enlargement, deformity and mass in the soft tissue suggestive of an encapsulated hematoma. Two weeks after the original injury, he was referred to the Post-Graduate Hospital, where he was *x*-rayed, but no treatment was instituted since operation was advised and refused.

I saw the patient nine weeks after the injury. A scar from the left scapula to the elbow joint remained as a result of a previous operation during childhood. (Fig. 1.) A plastic operation had been done in childhood for web fingers, with a resultant lymphangitis. The entire upper extremity was edematous and enlarged to about twice the size of the other. There was a circumscribed mass about 1 inch above the wrist, hard in consistency, and apparently attached to the bone. The area about the mass was inflamed and fluctuant.

X-rays showed extensive involvement of the distal half of the radius, with roughening of the periosteum and irregular decalcification. The principal changes were in the anterior medullary portion. There was an opaque mass 2 cm. distal from the bone, which might be a bony new growth and had the characteristics of an osteogenic sarcoma. (Fig. 2.)

After consultation it was decided to do a biopsy. Accordingly, on April 16, 1934, under local anesthesia, an incision was made through the skin and superficial fascia, and a great deal of fibrous tissue exposed. A portion of this mass was explored; it extended to the fascia between the radius and the ulna and was at-

tached to both bones. The major portion of the mass was enucleated and the periosteum of the bone was thus exposed. A sharp spicule of bone, which was protruding, was removed.

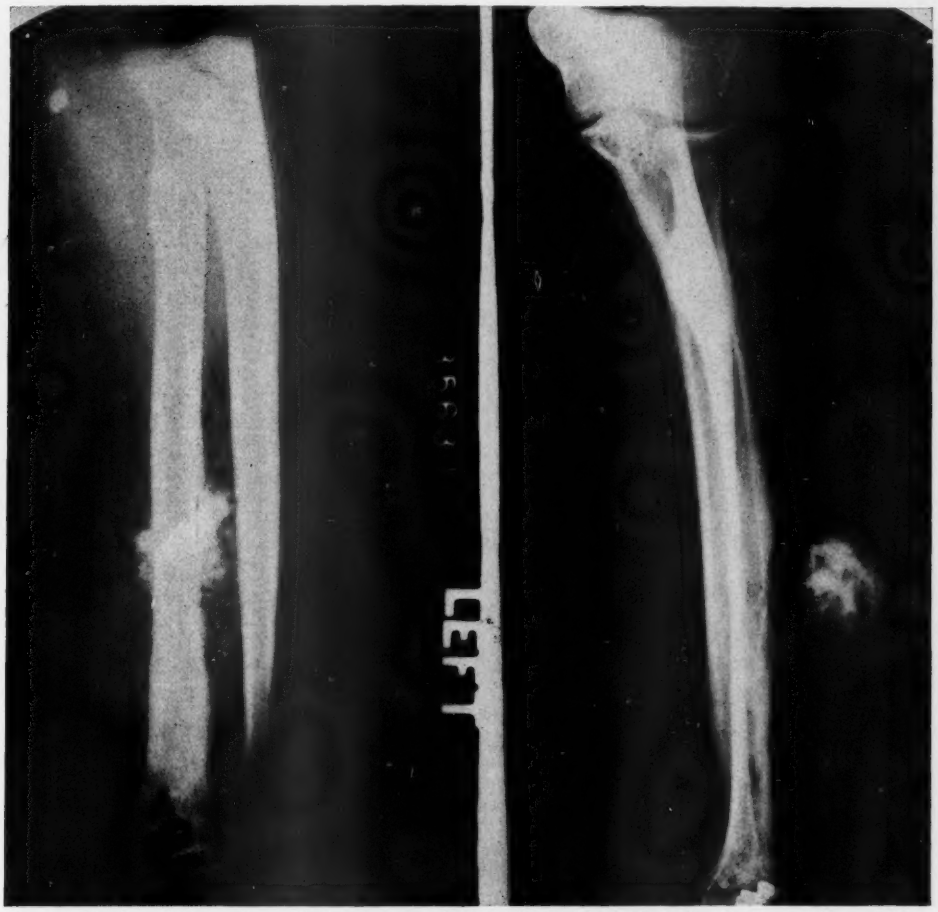

Fig. 2. Active destruction of the cortex of the distal third of shaft of the radius with no evidences of bone reaction here. There is, however, a discrete mass of new bone formation entirely separated from the cortex but within the tumor mass, probably due to secondary destruction.

Histologic examination showed a tumor consisting chiefly of fibrous spindle cells with multiple areas of necrosis. There were irregular fragments of bony tissue surrounded by fibrous tissue with evidence of necrosis. In one area of the fibrous tissue, there were a number of small spindle cells, which were moderately well differentiated but presented sarcomatous characteristics. The diagnosis of osteogenic sarcoma was given. (Fig. 3.)

Dr. Ewing was kind enough to reëxamine the tissue and agreed with the diagnosis. He also added that while some of the spindle cells were extraperiosteal in location, he thought that the growth was periosteal in origin. He advised Roentgen treatment because, while the tumor itself was radioresistant, very frequently small-cell tumors without definite bone formation yielded to persistent moderated and graduated doses of *x*-ray. It was his opinion that amputation was not justified and should be delayed until every method of conservative therapy had been instituted.

Dr. Alexander Fraser considered amputation the most feasible form of therapy since he felt that the growth was too highly differentiated to be sufficiently radiosensitive.

However, the plan suggested by Ewing was followed and the lesion was irradiated for several weeks, during which time reactions were carefully noted. There was no appreciable improvement in the neoplasm and amputation of the arm gave the only possible chance for cure.

On June 19, 1934, a high amputation of the left arm was done; the patient and his family were averse to a disarticulation. (Fig. 4.)

The patient made an uneventful recovery.

X-rays of the chest on August 15, 1934 were negative for metastasis. The patient was treated routinely until the wound was completely healed. He had gained more than 35 pounds about one year after amputation.

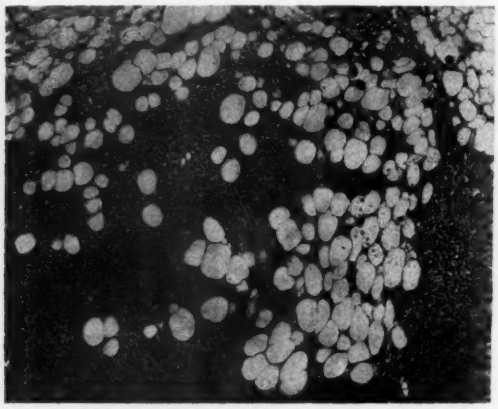

FIG. 3. Microsopic examination shows irregular fragments of bony tissue, surrounded by fibrous tissue, and evidence of necrosis. In an area in the fibrous tissue, there are a number of small spindle cells which are moderately well differentiated but present sarcomatous characteristics.

On January 2, 1936 the patient returned, complaining of pain in the chest, coughing and shortness of breath, all of three weeks' duration. *X*-ray examination disclosed massive pleural effusion on the left side, displacing the heart, trachea and mediastinum into the right chest.

On January 14, aspiration of the pleural cavity was performed, and about 600 c.c. of bloody fluid was removed. A histologic examination of the fluid revealed a hemorrhagic pleural exudate negative for tumor cells.

Aspiration was done five times, and the histologic reports at all times revealed a hemorrhagic pleural exudate without tumor cells or any identifying organism.

The patient was discharged from the hospital, and two later thorocenteses obtained fluid of the same character.

Coley's serum and additional *x*-ray therapy to the stump and lungs were instituted. At this time, metastatic deposits appeared disseminated in the scalp.

The patient finally succumbed on July 8, 1936, about two and one-half years after the original injury.

DISCUSSION

The conditions first propounded by Segond in Paris, and accepted by the courts and compensation bureaus in Europe and America that must be present to establish the relationship between an antecedent local trauma and subsequent development of a tumor, are: (*a*) the authenticity of the trauma; (*b*) sufficient importance or severity of the trauma; (*c*) reasonable evidence of the integrity of the body part prior to the injury; (*d*) correspondence of the tumor to the site of the injury; (*e*) date of appearance of the tumor not too remote from the time of the accident to be reasonably associated with it; (*f*) diagnosis established by clinical and roentgenologic evidence, supported if possible by microscopic examination.

FIG. 4. Gross specimen of amputated forearm. Near the area of ulceration there is invasion of fat, skin and tendon. There is a dense tumor nodule penetrating almost the entire thickness of the arm. Both the radius and ulna are smaller than normal. The radius is involved by the tumor and shows considerable destruction of the outer shell. There is definite marrow involvement. There are other small portions of bony spicules in some areas of the tumor.

We feel that the case reported is of special interest because of the hypothetical problems involved, opening fields for discussion of a practical nature. The six postulates listed above are generally accepted as formidable criteria. Evidence of integrity of the part prior to the injury is the question frequently productive of controversy in legal matters. The testimony must prove or disprove the presence of a neoplasm prior to injury. Certainly, a preëxisting tumor may be aggravated to more accelerated growth by trauma. Will an injury be adequate to promote malignant neoplasia in a part that has been devitalized over a period of many years?

We find no reference in the literature from which we may quote opinion. The case presented here gives substantial evidence of a part where the integrity had been disturbed for many years with old infection, lymphedema and stasis. I feel that severe trauma on this previously devitalized part was the immediate cause initiating a series of cellular changes which eventually led to a malignant bone tumor.

It is not amiss to suggest that future cases should be investigated more carefully to determine the integrity of the part. Careful history may yield clues of previous infections, injuries or degenerative processes which may have materially disturbed the integrity of the tissues.

SUMMARY AND CONCLUSION

This is a case of a young man, previously in excellent health, except for a congenital deformity of the left arm, forearm and hand, who sustained a direct injury to the arm and forearm. Two weeks after, a neoplasm of the forearm was found, which, upon histologic examination, proved to be osteogenic sarcoma. Radiation therapy proved futile, and was followed by a high amputation of the arm. Eighteen months later, metastases to the chest appeared. Death occurred two and one-half years after the time of injury.

I am of the opinion that there was a direct relationship between the injury and the subsequent development of the sarcoma.

It is worthy of mention that the causal relationship was established and upheld by the medical division of the New York State Department of Labor.

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A CASE OF INTERSTITIAL HERNIA*

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THIS report is prompted because of the relative infrequency of the condition to be described and the problem in diagnosis that confronted the members of both the medical and the surgical services who saw the case.

CASE REPORT

J. F., age 62, gravida 9, para 3, was admitted to the medical service on January 5, 1938, complaining of pain in the left iliac fossa of three days' duration. The patient had had an operation on the cervix in 1907, an appendectomy in 1910, fractures of both humerri sustained in an automobile accident, in 1928, a tonsillectomy in 1929, and a right herniorrhaphy in 1936. The previous history was otherwise essentially negative.

Three days before admission to the hospital, the patient took an alophen pill before retiring. On the following morning, she felt pain in the left lower quadrant which was not severe but was persistent and remained localized.

Examination demonstrated a mass shaped like a sausage in the left lower quadrant, parallel to Poupart's ligament. The mass was tender to touch. The general condition of the patient was excellent. The hemoglobin was 78 per cent; red blood cells numbered 3,750,000; the white blood cells numbered 9,200, of which 62 per cent were polymorphonuclears. Stool examinations were negative for blood. The urine was normal, the temperature and pulse not elevated, and *x*-ray and sigmoidoscopic studies negative. The original opinion of the visiting staff was that there was an intra-abdominal tumor, probably a neoplasm in the sigmoid.

On January 8, 1938, the patient was operated upon under spinal anesthesia. An oblique incision, parallel to the long axis of the mass, was made in the left lower quadrant (the operator feeling that the most likely diagnosis was a tumor in the abdominal wall). The fibers of the external oblique were divided directly over the palpable tumor. On retracting the edges of the external oblique incision, a banana-shaped mass, about 3 inches long, was found, lying below the external oblique and between it and the internal oblique. The mass was adherent to the surrounding structures and had a bluish-green color. It extended from the general neighborhood of the internal ring upwards and outwards. Tracing the mass downwards seemed to show it continuous with and disappearing into the internal ring. Splitting of the covering disclosed that it was a hernial sac containing adherent gangrenous omentum. The omentum was freed and was resected in healthy tissue, after which the sac was treated by high ligation and ablation, as is customary in a hernioplasty. The operation was completed by an Andrew Ferguson herniorrhaphy. No other sac running towards the labia or along the round ligament was noted.

The postoperative diagnosis was interstitial hernia.

The postoperative course was uneventful except for a small hematoma in the lower angle of the wound.

DISCUSSION

The generally accepted views regarding the etiology of interstitial hernias is that an ectopic testicle diverts the hernial sac from its normal descent into the scrotum. Because MacReady, Gobel, and Lower and Hicken report a majority of the collected cases in their series associated with an ectopic testicle, this must be considered as an important factor. That this is not the only factor, however, or even an essential one, is readily appreciated when one uncovers the numerous case reports of interstitial hernia in men with normally placed testicles, and also in women. In many of the reported cases of this type of hernia in males with normally placed testicles, a constricted external ring, congenital or acquired in origin, has been found. There is

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also a case on record of a hydrocele in the Canal of Nuck, evidently the obstructing factor in the causation of the interstitial hernia. In our case, as in Lower and Hicken's two cases, no obstruction was encountered in the inguinal canal or at the external inguinal ring. Nevertheless, it appears to us that some obstructive factor must in large measure be responsible for the occurrence of this hernia.

Interstitial hernia is one of the three types of a group of abnormal hernias in the inguinal region known under the heading of interparietal hernia. The latter may be defined as a group of unusual inguinal hernias in which the sac has failed to follow the course of the spermatic cord and has spread itself out between the various layers of the abdominal parietes. The interstitial variety, with which we are chiefly concerned in this paper, includes those hernias in which the sac lies either between the transversalis fascia and transversalis muscle, or between the internal oblique muscle and the external oblique aponeurosis. There is still another type of interstitial hernia in which the sac lies within the fibers of the internal oblique muscles. This, however, is a very rare condition. Moynihan denies the existence of any variety of interstitial hernia other than that in which the sac is found between the oblique muscles. Authentic cases, however, of other varieties have been reported by Gobel and Coley and Sultan.

The most common variety of interstitial hernia, as mentioned previously, is that in which the sac lies between the two oblique muscles. The case here reported, those of Lower and Hicken and that of Biegler were in this category. The probable explanation for this is purely a physical one and lies in the greater potential space and loose connective tissue between the two oblique muscles.

Although Moynihan, Halstead and Watson contend that biolocular sacs must be present in these types of hernias, our case presented only one sac, and about one-third of the cases reported in the literature had monolocular sacs.

Intestinal obstruction is usually encountered as the outstanding symptom. There were ninety-seven incarcerations in Gobel's 115 cases. Intestinal obstruction was absent in the cases of Lower and Hicken, Biegler, and in our own. The sac in our case, however, despite the absence of obstructive symptoms, contained gangrenous omentum.

In Biegler's and in our own case, a palpable mass above Poupart's ligament was found. In one of Lower and Hicken's cases, there was a swelling above the external inguinal ring.

The diagnosis should not be difficult in those cases presenting obstructive symptoms associated with an abnormally placed testicle and a palpable mass above Poupart's ligament. In other cases without the above combination of symptoms and signs, the diagnosis may be difficult or impossible. In the presence of a mass with pain alone, a differential diagnosis between diverticulum of the intestine, neoplasm of the intestine, tumor of the abdominal wall and interstitial hernia may be difficult, as our experience in this case shows. The diagnosis, however, should be suspected if the opposite side presents a hernia of the usual variety or there is a history of a previous herniorrhaphy, even though the patient may present only a mass and pain in one of the lower quadrants. Biegler's patient presented an ordinary inguinal hernia on the opposite side and ours gave the history of a herniorrhaphy performed two years previously on the opposite side.

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ON EXTRARENAL RETROPERITONEAL TUMORS*

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SINCE reporting a case of ganglioneuroma in 1929, which was retroperitoneal and extrarenal, we have encountered five other tumors so characterized by their location. There were three neuroblastomata in children, one leiomyosarcoma in an elderly woman, and one metastatic tumor in a middle aged male which arose from a tiny embryoma of the testis.

In all cases urologic structures were involved and all cases except the second had complete preoperative urologic examinations. The diagnostic difficulties in the fourth and the fifth case deserve particular attention.

The surgical difficulties were all due to blood supply control. The aorta was intimately involved in the third case, so much so that removal was impossible. It was also involved in the fifth case, but not so intimately. The renal vessels were the main source of blood supply in the first and the fifth case making the operations fairly simple. In the second case branches from the internal iliac artery supplied the tumor. The vena cava was invaded in the fourth case, thereby causing inoperability.

The surgical approach was transperitoneal in the first three cases and retroperitoneal in the last two.

One of the patients died shortly after the operation and one died two months later. The other three are alive and apparently well today.

CASE REPORTS

CASE I. The patient was a female child, aged 4 months. She was brought to the hospital because of vomiting, loss of weight, and mass in the abdomen noticed for the preceding three weeks. A large, round hard mass was found in the left side of the abdomen, extending from the costal margin to the iliac crest, and from the midline to the loin. The mass was painless and movable.

Cystoscopy showed P.S.P. function from both sides in eight minutes. A left pyelogram showed displacement of the left ureter medially and the left kidney high up in the abdomen. The left kidney pelvis and the calices showed crowding. (Fig. 1.) The chest and the long bones were roentgenologically negative.

An operation for removal of the mass was undertaken. Through a T-shaped abdominal incision and a transperitoneal approach it was possible to free the tumor completely and to determine its attachment to the lower part of the left kidney capsule. Due to the blood supply it was necessary to remove the kidney along with the tumor.

On pathologic examination the tumor proved to be 9 cm. in each of its three diameters and to be attached to the lower and mesial surface of the kidney capsule. The kidney itself was not involved. The histologic report was as follows: "The tumor is divided into lobules of varying size and shape, separated by connective tissue bands. The cells resemble medullary neural epithelium; they are for the most part small and of varying shape. Many of them produce glial fibers and in some foci well formed rosettes are seen. Aside from the rosettes the general architecture is that of strands of primitive neural epithelium found in some teratomata." (Fig. 2.) A diagnosis of neuroblastoma was made.

The postoperative recovery of the patient was uneventful and the child is reported to be alive and well four years after operation.

CASE II. A female child, aged 18 months, was brought to the hospital with the chief complaint of a mass in the lower abdomen which had been noticed for eight months and which had been slowly increasing in size.

Examination revealed a large, hard, nodular tumor in the right hypogastrium. It was very slightly movable and not painful; on rectal examination it appeared fixed to the right

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sacroiliac synchondrosis. Cystoscopy was not undertaken, because the tumor could be outlined so definitely by palpation. The preoperative diagnosis was retroperitoneal neurocytoma.

Fig. 1. Displacement of left ureter and left kidney.

The operative approach was through an incision slightly to the right of the midline. The posterior peritoneum was incised medially to the right ureter which was anterior to the tumor. Dissection was fairly easy at first; the external iliac vessels and the ureter were easily separated and the tumor was then found to emerge from the first sacral foramen and to derive its blood supply from the internal iliac or hypogastric artery. It was necessary to cut numerous vessels arising from this artery and also several nerve bundles which were thought to come from S 1 and 2. The operation was very bloody and removal of the tumor was not thought to be complete, small portions of it apparently remaining in the first sacral foramen.

The tumor measured 7 by 8 by 4 cm. and had a thin capsule. On cross section it showed grayish-white nodules in a dark red stroma.

Sections showed a tumor made up of small, deep staining cells, arranged in nests or lobules which were separated by a fibrous stroma. The cell bodies were rather widely separated by masses of glial fibers, which were very delicate and cobweb-like in texture. In some areas the tumor closely resembled cerebral tissue with occasional ganglionic cells. The growth did not appear to be very malignant, but was definitely of neurocytic origin, a neuroblastoma. (Fig. 3.)

The only postoperative complication was a foot drop on the right; the neurologic consultant thought this due to injuries to the fifth lumbar and the first sacral nerves. The child is well today over four years after the operation. Apparently there is no recurrence and the foot drop has disappeared.

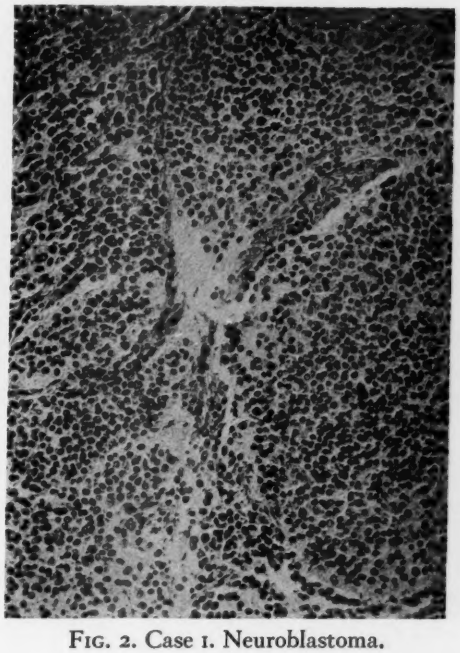

Fig. 2. Case 1. Neuroblastoma.

Case III. This was a female child, aged 26 months. The chief complaint was an abdominal swelling which had been noticed for five weeks and which was growing rapidly.

Examination showed a huge, nodular, firm, and not tender mass which almost filled the abdomen and which was not movable. It was more prominent in the midline, but extended well into both flanks. Roentgen plates showed the bowels displaced by the mass and an enlarged liver. Chest plates showed a marked mediastinal shadow protruding into the left lung. The long bones were negative.

On cystoscopy the P.S.P. function on the right was eight minutes and on the left fourteen. Pyelogram demonstrated marked displacement of both ureters laterally and compression of both ureters. (Fig. 4.)

A transperitoneal exploratory operation was undertaken in spite of the apparent hopelessness of the case. The mass was found to be retroperitoneal and extended from the level of the gastrohepatic omentum down to the bifurcation of the common iliac arteries. The ureters as well as the kidneys were pressed laterally by the mass. There were many enlarged lymph glands in front of the tumor. Since removal was impossible, two of the en-

larged lymph glands were removed for histologic examination and the abdomen was closed.

The histologic examination of the lymph

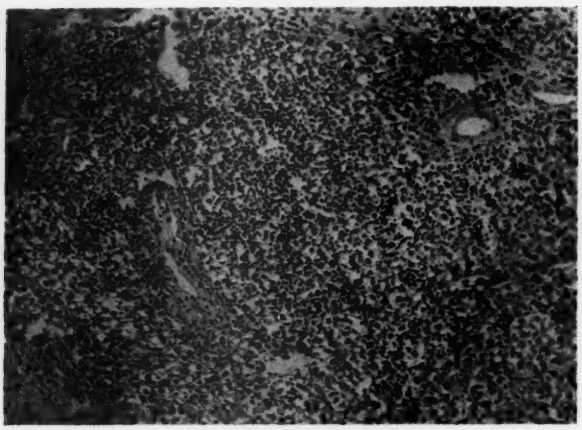

Fig. 3. Case II. Neuroblastoma.

glands showed the nodes replaced by tumor tissue, composed of small cells with deeply stained round vesicular nuclei. The cell bodies were ill defined and consisted of narrow rims of cytoplasm which streamed into fine processes, forming an intercellular glial network. Occasionally the cells were arranged in small rosettes. Numerous mitotic figures were present. The

Fig. 4. Case III. Displacement of both ureters.

tumor, a neuroblastoma, was highly vascular and appeared to be very malignant. (Fig. 5.)

The child had an uneventful postoperative course, but the tumor continued to grow and the patient died two months after the exploratory operation.

Case IV. This woman, 70 years of age, had as chief complaint a swelling in the right side of the abdomen which had been present for eleven months and which caused a great deal of pain. She stated that the mass has been

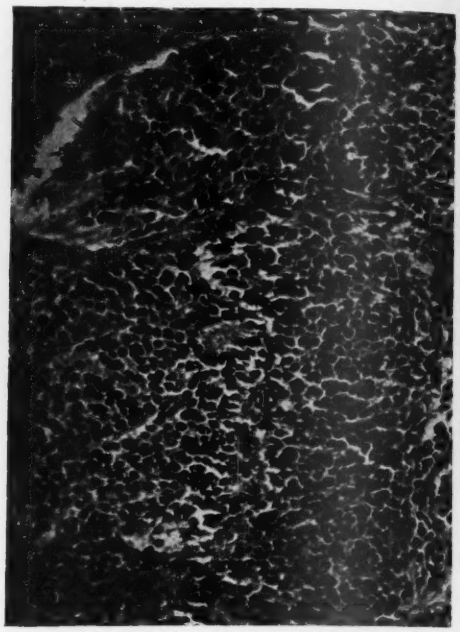

Fig. 5. Case III. Neuroblastoma.

growing slowly, but that lately the pain had been getting very much worse. Her general condition was poor and she had suffered for many years from hypothyroidism and adiposity.

Examination showed a huge mass in the right side of the abdomen. It was irregular in outline, not movable and quite tender. Distinct fluctuation could be felt. As far as we could tell it extended from the costal margin to somewhere below the iliac crest and seemed to be retroperitoneal; intestines could be palpated in front of the mass.

A barium meal merely showed displacement of the bowel by the mass and a vaginal examination seemed to rule out tumor arising from the pelvic organs.

On cystoscopy a steady drip of hazy urine was observed to come from the right kidney, which continued during observation, namely for 30 minutes. P.S.P. appeared from the left in four minutes, but none appeared from the right during the period of observation. A right pyelogram with 20 c.c. of contrast medium showed a huge misshapen pelvis, but no calices. The outline of the kidney could not be discerned.

Because of the pain and in spite of the patient's poor general condition, operation was undertaken for drainage of the hydronephrotic sac. Under local anesthesia the lumbar ab-

dominal wall was dissected down to the mass and 1,500 c.c. of a light brown watery fluid drained off. At the end of the drainage blood

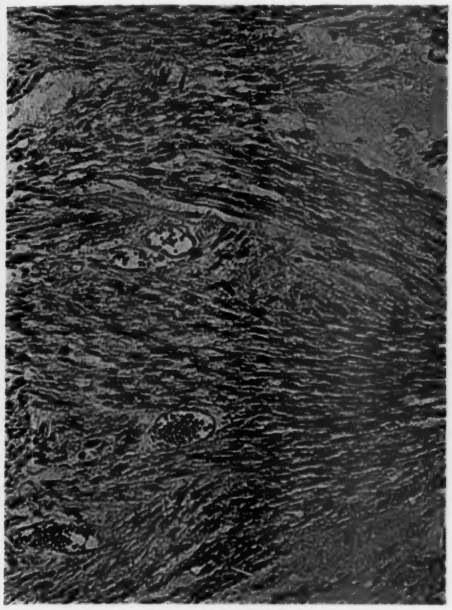

Fig. 6. Case iv. Leiomyosarcoma.

appeared through the trocar. A gauze pack and compression seemed to control the bleeding, but the patient died a few hours later.

The post-mortem examination revealed a very complex state of affairs. The nucleus of the pathologic changes found was a large hard nodular tumor in the right lumbar gutter. It extended from the level of the second lumbar vertebra to the upper margin of the sacrum. Its upper pole was attached to the right kidney and had invaded and compressed the vena cava. The compression of the vena cava had produced numerous huge varices of the veins of the abdominal wall. The fluid drained off during the operation had been in a retroperitoneal, but extrarenal cavity. It is probable that it formed on the basis of an old hematoma. The operative hemorrhage was due to a ruptured varix. The right kidney was totally hydronephrotic, and the right ureter was compressed along its upper abdominal portion by the tumor.

Histologic examination showed large spindle-shaped cells arranged in broad interlacing bundles. The nuclei were generally oval and vesicular, but many of them were very large, hyperchromatic or showing mitotic figures. Occasional multinuclear forms were seen. The cell bodies were large and stained yellow by the Van Gieson method. A scanty connective tissue stroma distributed throughout the growth carried a fairly abundant vascular supply. The

Fig. 7. Case v. Note left hydronephrosis and compression of left ureter.

tissue was rather edematous. Diagnosis was leiomyosarcoma. (Fig. 6.)

Case v. The patient was a male 39 years of age whose chief complaint was pain in the region of the left kidney of three months' duration. His abdomen was too fat to allow accurate palpation. A pyelogram showed marked

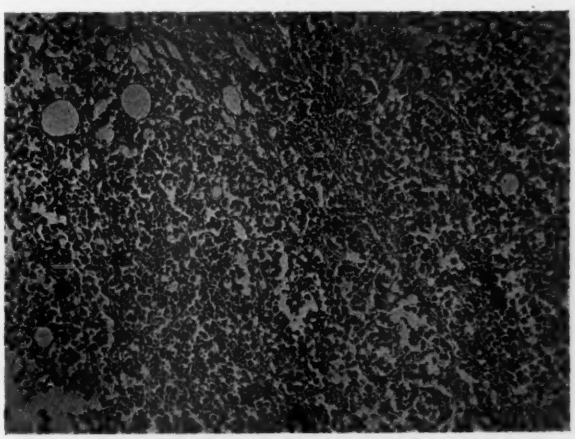

Fig. 8. Case v. Metastatic embryonal carcinoma of testis.

hydronephrosis of the left kidney with decreased function and marked and persistent compression of the left ureter below the kidney pelvis. (Fig. 7.) On operation a hydronephrotic kidney was found. The compression of the ureter was due to an extrarenal tumor which extended from the pedicle of the kidney down to the third lumbar vertebra. The tumor was smooth, white and firm. It was densely attached to the aorta. Removal of the kidney and the tumor was undertaken, but it could not be

separated from the aorta wall. A small strip of tumor tissue had to be left behind, measuring about 7 cm. in length.

The tumor was composed of broad sheets and nests of rounded or polyhedral cells, separated by a delicate stroma network and occasional broader trabecular bands. The tumor cells were of fairly uniform size and shape. Their nuclei were rounded and vesicular; the cell bodies were pale and vacuolated. In the stroma were abundant lymphocytes. (Fig. 8.) The diagnosis was metastatic tumor probably arising from an embryonal carcinoma of the testicle.

Examination of the testis showed no increase in size and no nodules, but the left epididymis was distinctly thickened and indurated. Nevertheless, it was decided to remove the testicle. Even after the testis was taken out of the scrotum it was impossible to discern any tumor, and only after removal and cross-section, was a pea sized, yellow tumor found in the center of the testicle. Histologic sections of this tumor showed marked central necrosis, but the marginal portions showed the same histologic picture as the metastatic tumor.

The patient made an uneventful recovery, and was then treated with deep Roentgen rays. He is alive and apparently well two years after the operation.

SUMMARY

Three cases of retroperitoneal neuroblastomata in children are reported. Apparently they differ widely in their degree of malignancy. Removal of such tumors should be attempted since cure has been effected in two of the three cases reported here. The second case is particularly remarkable, because removal of the tumor seemed to be incomplete and yet the child made a complete recovery.

One case of retroperitoneal leimyosarcoma is reported which is remarkable on account of the complexity of the pathologic findings.

One case of metastatic testicular tumor in the retroperitoneal space is reported. It tends to corroborate the fact that such tumors can be cured by deep roentgen therapy.

Grateful acknowledgment is due to Drs. D. D. Davis and W. H. Field for some of the clinical material reported here and to Dr. J. A. deVeer for the careful analysis of the pathologic findings.

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DIDELPHYS IN SISTERS

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E. T., white, age 12, was admitted to the Greenville General Hospital in September, 1937, complaining of vaginal bleeding. Her periods began at 11 years of age. She menstruated regularly for four months, missed two months, then began bleeding, and continued to bleed until admission. She was well developed, apparently 16 years old. Except for pallor and tenderness in the lower half of the abdomen, the general physical examination was negative. A possible pregnancy was excluded by a negative Friedman test. On examination under anesthesia, there was no abnormality of cervix or vagina. A uterine sound introduced through the cervix, passed to the left. It could not be directed to the right. There was a mass about 4 cm. in diameter, round, to the right, and close to the body of the uterus, which was inclined to the left. Double uterus and ovarian cyst were considered as possible diagnoses. Laparotomy at a later date was decided upon.

When the abdomen was opened, the pelvic structures appeared as in Figure 1. Removal of the mass to the right of the uterus was attempted. This had partly succeeded when we encountered bleeding which we could not control. It was decided to remove the uterus supravaginally to check the bleeding. This was done, but it was necessary also to pack the wound before bleeding ceased. The left ovary was retained. The wound was closed around the packing.

The patient lost a great deal of blood. Hard work by the internes and immediate transfusion, repeated the following day, saved her life. She finally recovered, and left the hospital with the wound nearly healed. When seen three months later, she was in good health. It was explained to the mother that the child could not menstruate and could never bear children.

The pathologist reported that there were two bodies to the uterus, one tube attachment to each. Several pieces of tissue were removed from a cyst wall and section showed a denuded endometrium. Microscopically, the cyst wall had fibromuscular stroma, covered by stratified squamous epithelium. This was denuded, and had granulation tissue. The cyst contained simple columnar epithelium. The tissue suggested congenital misplaced vaginal structure.

The left cornu had been curetted shortly before operation. The "denuded" areas were in this curetted cornu.

Four months later, in January, 1938, the patient's sister, A. T., aged 14, was admitted. She complained of pain in the lower abdomen for the past three months and cessation of periods. She had not been so uncomfortable since periods ceased as she was while they had been present. Menstruation had begun at 11 years of age. With onset of periods, there was nausea, vomiting, and confinement to bed, often for a week.

This patient also was very well developed for her age. There was nothing unusual in the general physical examination.

Under anesthesia, the vagina and the cervix were examined and found normal. A uterine sound passed to the left into the uterus. A mass slightly larger than the uterus lay closely adherent to the right side. The sound could not be introduced into this mass. We made a diagnosis of double uterus, and immediately opened the abdomen. (Fig. 2.)

The right cornu was larger and softer than the left and was removed with its tube. No communication could be found between its cavity and the cervix or vagina. The epithelium remaining was curetted and cauterized with iodine. The raw surfaces were peritonized, the appendix removed, and the abdomen closed. Recovery was uneventful. The cornu of the uterus was hypertrophied, and showed a thickened, softened, and congested endometrium in longitudinal folds, as if it had been distended.

In September, 1938 both patients were in good condition.

It is unusual for double uterus to occur in sisters. An interesting feature of the history is that their mother is the second wife, and that among four children from the father's first marriage there was no complaint of "female trouble." A visual and bimanual pelvic examination of these pa-

tients' mother showed normal vagina and cervix. The abdominal wall was so thick that nothing could be made out bimanually.

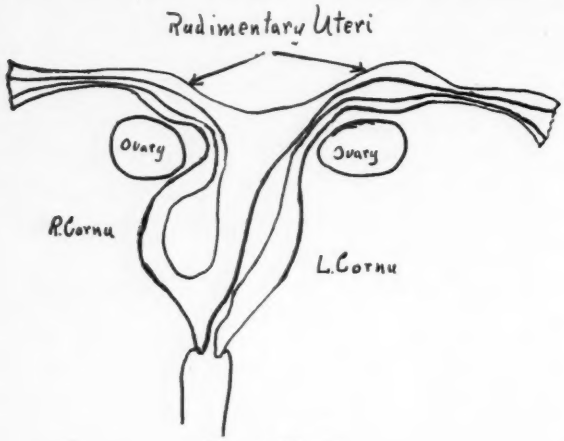
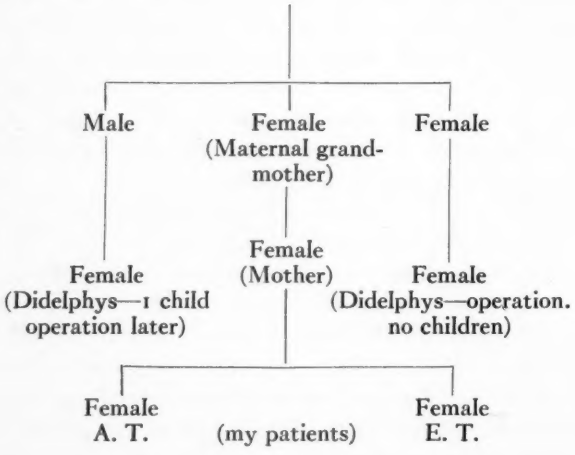


Fig. 1. Case I.

An interesting family history was later obtained from the mother who reported that two maternal cousins had double uterus, proved by operation. One of these cousins had borne a child before she was operated upon.



Conservative treatment of didelphys is generally agreed upon among gynecologists. Many such patients have had normal pregnancies. Some patients have had a normal pregnancy in one horn and a later pregnancy in the other horn. Some cases have been discovered only at operation, the previous history giving no hint of unusual conditions. Nearly 50 per cent of pregnancies occurring in such patients terminate in miscarriage.

In the first case, had we been fortunate

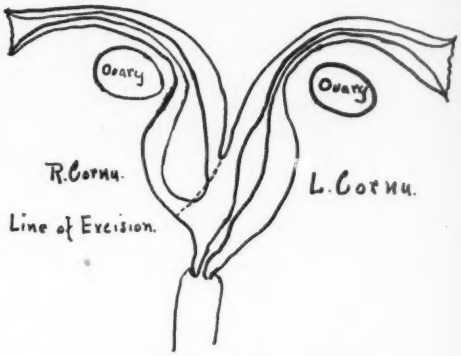


Fig. 2. Case II.

enough to remove the right cornu, it is interesting to speculate on the possibilities of pregnancy and its outcome. The small masses at the uterine ends of the tubes were undoubtedly rudimentary uteri, also supernumerary. Fertilization having occurred in the tube, the ovum would most likely have lodged in this structure. If it matured, Cesarean section would probably have been necessary.

In the second case, one apparently normal horn of the uterus remains with its tube and two ovaries. The possibility of pregnancy is scarcely less than in a normal uterus one of whose tubes has been removed.

SUMMARY

An instance of didelphys in sisters is presented. Probably other such cases are known but have not been published. Frequently the condition is unrecognized. Discovery is not an indication for operation unless symptoms demand it. If operation is done, the surgeon should be as conservative as possible. A secondary more radical operation is preferable to depriving the patient of her child-bearing function unless this is clearly indicated. It is better to allow the patient a test of pregnancy. She or her family should be told of the condition; and if pregnancy occurs, she should be kept under close observation.

MALIGNANCY OF THE ANUS, RECTUM AND SIGMOID COLON IN THE YOUNG*

WITH REPORT OF A CASE AT FOUR AND A HALF YEARS

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UNTIL the etiology of cancer is known and a specific treatment developed; while our research colleagues continue their own experiments and evaluate the numerous hypotheses and therapies suggested by others; the medical profession at large must continue the education of the laity in regard to malignancy, and we, the clinicians, must content ourselves with endeavoring to put into as many hands as possible the only weapon at present at our command—early diagnosis.

Carcinoma of the rectum and sigmoid colon in adults is rather common, comprising approximately 12 per cent[104] of all malignant neoplasms. This figure is sufficiently high to arouse every physician to suspect a malignant process when a history of bleeding or change in bowel habit is given by the patient. In youth, however, the occurrence of malignancy is computed at 4 per cent,[103] and of this only a small proportion can be assigned to the lower bowel. Nevertheless, the fact that even a relatively small number of tumors have been and continue to be found in the rectum and sigmoid in children should prompt us to suspect malignancy at any age, even though the clinical train of symptoms is obscure or indefinite.

Fowler,[31] in an excellent article on malignancy in persons under 26 years of age, noted that 18.7 per cent of neoplasms were in the large bowel, and of these 12.5 per cent were located in the rectum and rectosigmoid. A review of several large series shows that cancer of the distal bowel is infrequent in youth and early adult life. (Table 1.)

TABLE 1

Investigator	Number of Cases	Percentage below 30 Years	Percentage below 20 Years
Matzen[67]	8054	.....	.003
Pennington[82]	7174	.032	.005
Rosenberg[95]	4048	.....	.002
Bacon[5]	1995	5.4	.004
Rankin[92]	1452	3.85	
Janusz[48]	831	.....	.0024
Wildholz[114]	563	1.95	
Glazer[35]	527	3.5	
Lynch[60]	491	7.0	
Passler[80]	282	1.0	
Schreiner[101]	210	2.6	
Brindley[11]	100	2.0	
Fitzwilliams[29]	....	11.0	4.0

In spite of such evidence, however, we should be ever mindful of the possibility of such conditions in youth. Just recently a case came under our own observation, which is reported here.

CASE REPORT

H. L., a lad of 4 years, 7 months, was brought to the Children's Clinic of the Philadelphia General Hospital because of bleeding and protrusion from the bowel. As related by the child's mother, about one and a half years before blood was first noticed in the toilet bowl after a bowel movement. During the ensuing four months the stools were streaked with blood about once a week; but for the past year bleeding had

* From the Surgical Service of Dr. J. C. Howell, Department of Radiology, Philadelphia General Hospital. Read before the Proctologic Society of the Graduate Hospital, University of Pennsylvania, Philadelphia, February 9, 1938.

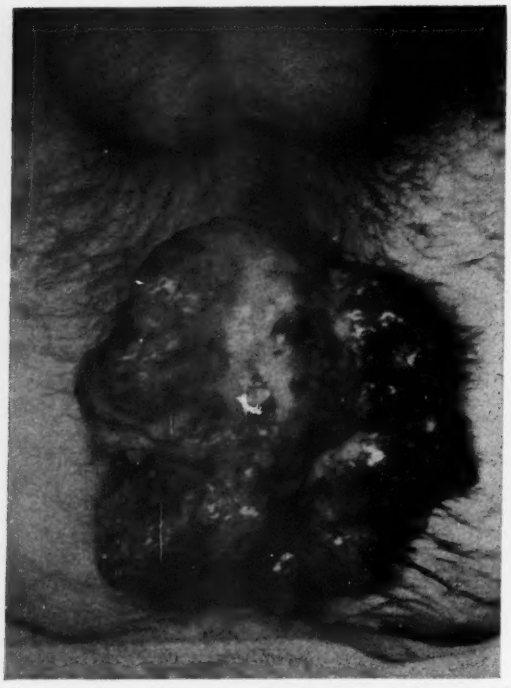
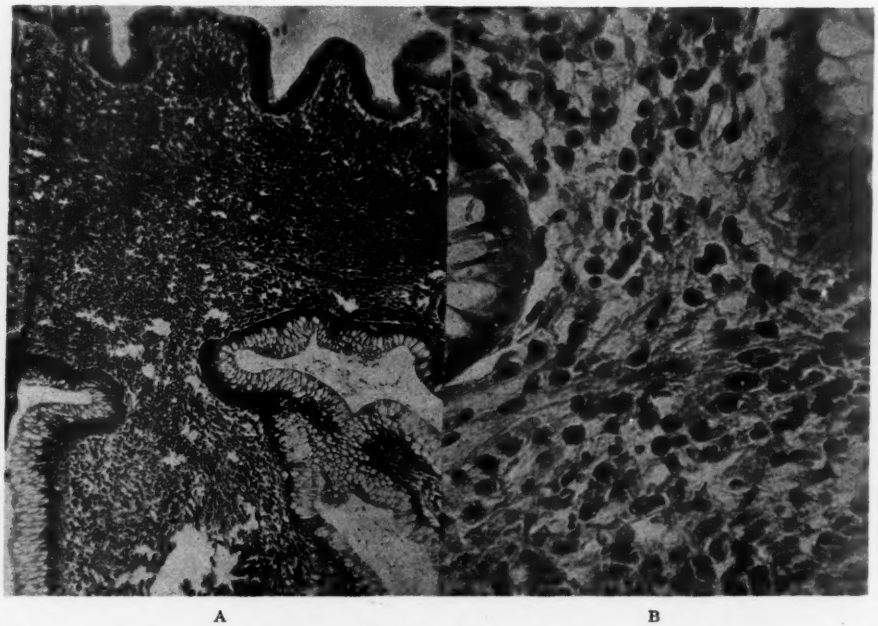


FIG. 1. Fungating protruding adenocarcinoma of rectum in a young man of 17 years. (Courtesy of C. F. Martin.)

FIG. 2 Boy 4 years and 7 months old, with early carcinoma of rectum.



A B

FIG. 3. A, low power section through polypoid growth of rectum. Note hyperplasia of acinar epithelium and inflammatory changes in stroma. B, high power magnification of same specimen, showing poorly defined basement membrane. Some of glandular components are lined by columnar goblet cells, presenting normal maintenance of nuclear polarity, while a few exhibit hyperchromatic cell lining accentuated by definite loss of nuclear polarity assuming pseudostratified and stratified arrangement, mitotic figures being frequently encountered.

been more or less constant following each defecation. About four weeks previous, approximately a teaspoonful of fresh blood was passed, and this situation had persisted until the time of admission to the Clinic.

When blood was first noticed, the little patient was examined by his parents, who found a blood-streaked mass about the size of the rubber tip of an ordinary lead pencil protruding from the anus. The small mass was pushed back into the rectum with a finger covered with toilet paper and vaselin. No pain had been experienced by the child at any time.

In his history, mumps, measles, chicken pox, and whooping cough were cited. Both the father and mother were living and well, aged 32 and 29 respectively. No family history of cancer was obtained.

The patient was referred to the Proctoscopic Clinic, where a polypoid growth about the size of the distal phalanx of the index finger was found. Dr. Philip R. Custer, pathologist of the Philadelphia General Hospital reported that this was a firm, encapsulated mass, 15 by 12 by 8 mm., accompanied by a small piece of firm, pale gray tissue. On cross section the mass appeared to be composed of mottled gray and purple tissue.

The surface of the polyp was denuded of epithelium and underlaid by a zone of acute and chronic inflammation. Some of the glandular components were lined by columnar goblet cells, showing normal maintenance of nuclear polarity. A few, however, exhibited hyperchromatic cell lining accentuated by definite loss of nuclear polarity, assuming pseudostratified and stratified arrangement. Mitotic figures were frequently encountered. Dr. Custer was inclined to regard these as evidence of early malignancy. Two other pathologists have confirmed this diagnosis.

The patient was admitted to the children's department June 21, 1937 and a radiologic consultation was requested. Digital and proctosigmoidoscopic examination elicited no pathology; yet, because the child was obstreperous, reëxamination under vinethene was considered expedient. This was also negative.

Dr. B. Holmes three days later reported that fluoroscopic examination of the rectum and sigmoid showed no pathologic lesion. The large intestine was apparently normal. A contrast enema showed the pelvic, transverse and ascending colon well filled with air, barium being present only in the rectum, a part of the sigmoid and cecum. There was no evidence of polyposis. This study was repeated on June 28 and the findings essentially confirmed.

The urine examination was negative. Blood count showed: erythrocytes, 4,180,000; leucocytes, 7,700; hemoglobin, 78 per cent; differential: polymorphonuclear leucocytes, 50 per cent; lymphocytes, 48 per cent; transitionals, 2 per cent. Blood sugar was 87 mg. per 100 c.c.; blood urea, 14 mg. per 100 c.c. The Kahn test was negative.

The patient was discharged from the hospital on June 30, 1937 but has been followed in the Radiologic Clinic periodically. There has been no evidence of recurrence, and the child has gained weight.

A review of the literature citing proved cases of anal, rectal, and sigmoidal malignancy in patients 19 or under has been condensed into Chart 1.

Warthim[112] has mentioned that malignant tumors are relatively rare before puberty, but that there is a steadily ascending line of occurrence from childhood on to middle age, both sarcomata and carcinomata appearing with about equal frequency. In this series of collected cases it will be noted that the largest number for any age below 12 is four, but that above 12 the smallest number of cases for any age is at least twice that number. It is also to be noted that there is an increasing number with each ascending age group. This table shows that there are sixty-five males and twenty-eight females, a ratio of 2.32 to 1. The site was in the rectum in eighty-five cases, the sigmoid in nineteen, the anus in one, and unstated in sixteen.

The symptoms as recorded in thirty-five cases are given in Table II. This is in contrast to the symptoms in adults as reported by one of us.[4] (Table III.)

Table II

Abdominal pain	17
Bleeding	9
Constipation	7
Rectal pain	4
Diarrhea	3
Lumbar pain	1
Loss of weight	1

CHART I

	Age	Sex	Location	Symptoms	Duration of Symptoms	Duration of Life	Type of Tumor
Ahfeld[1]	Newborn monster	..	Rectum				
Clar[15]	3	M	Sigmoid				
Lasnier[54]	3	..	Rectum	.....	.....	.....	Epithelioma
Ullhorn[111]	3½	M	Sigmoid				
Goodman[37]	4	F	Sigmoid	Chronic constipation	4 weeks	.....	Lymphosarcoma
Duncan[28]	5	M	Rectum				
Ullhorn[111]	5	M	Rectum				
Lockhart-Mummery[58]	8	..	Rectum				
Philipp[84]	9	M					
Steiner[109]	9	M	Sigmoid	Pain in abdomen	2 weeks		
Leijer[57]	9	M	Sigmoid	Constipation	11 days		
Parkinson[79]	9	M	Rectum	.....	6 months		
Rowntree[98]	10	M	Rectum	.....	.....	.....	Sarcoma
Stern[110]	11	F	Rectum	Abdominal pain			
Smith[105]	11	M	Rectum	Bleeding	6 months	.....	Adenocarcinoma
Bethe[8]	11	..	Rectum				
Rankin[91]	11	..	Rectum	.....	.....	.....	Lymphosarcoma
May[68]	12	F	Rectum				
Paultauf[81]	12	F	Sigmoid				
Garrard[34]	12	M	Sigmoid	Constipation			
Zuppinger[116]	12	F	Sigmoid	Abdominal pain	3 months		
Parkinson[78]	12	F	Rectum	Abdominal pain	.....	.....	Adenocarcinoma
Marchand[63]	12	F	Anus	Rectal pain	9 months		
Cummings[21]	12	M	Rectum				
Busche[12]	12	M	Rectum	Pain in rectum			
Mayo[69]	12	M	Rectum				
Philipp[84]	12	F					
Milne[72]	12	M	Rectum	Intestinal obstruction	Few days	.....	Colloid
Mandl[62]	12	..	Rectum				
Nothnagel[74]	12	F	Sigmoid				
Jordan and Chamberlain[49]	13	M	Rectum	Diarrhea	21 months	.....	Adenocarcinoma
Czerny[22]	13	M	Rectum	.....	18 months	.....	Adenocarcinoma
Allingham[2]	13	M	Rectum				
Gurlt[41]	13	M	Rectum				
Gowland[38]	13	M	Rectum				
Kupfelere[52]	13	F	Rectum				
Raiford and Buttles[89]	13	F	Rectum	Lumbar pain	4 weeks	.....	Colloid carcinoma
Babcock[3]	13	F	Rectum				
DeSaive[25]	13	..	Rectum				
Bottin[10]	13	F	Rectum				
Rostoker[97]	14	..	Rectum				
Schneider[99]	14						
Philipp[84]	14	M					
Mayo-Robson[70]	14	..	Rectum				
Caird[13]	14						
Davis[24]	14	M	.....	.....	.....	.....	Adenocarcinoma
Mandelung[61]	14	..	Sigmoid				
Olmsted[76]	14	M	Sigmoid	Abdominal pain and diarrhea	4 months	.....	Adenocarcinoma
Porter[87]	14						
Cripps[19]	14	M	Rectum	.....	2 months	3 months	Adenocarcinoma

CHART I (*Continued*)

	Age	Sex	Location	Symptoms	Duration of Symptoms	Duration of Life	Type of Tumor
Israel[46]	14	M					
Michauz[71]	15	M	Rectum	.....	.....	.....	Colloid
Wilde[113]	15	M	Rectum				
Marsh[64]	15	M	Rectum	Abdominal pain	6 months	.....	Adenocarcinoma
Bernouille[7]	15	M	Rectum				
Clogg[17]	15	..	Rectum				
Porter[87]	15						
Godin[36]	15	..	Sigmoid	.....	.....	.....	Scirrhous carcinoma
Yeomans[115]	15	M	Rectum				
Gant[33]	15	F	Rectum				
Fowler, R. H.[32]	15	M	Rectum	Pain in rectum and abdomen	9 months	.....	Adenocarcinoma
Philipp[84]	15	M					
Rome[94]	15	M	Rectum	Abdominal pain	1 month	.....	Adenocarcinoma
Schneider[99]	15						
Rostoker[97]	15						
Kaleniewicz[50]	16	M	Sigmoid				
de la Camp[53]	16	..	Rectum				
de la Camp[53]	16	..	Rectum				
Gant[33]	16	M	Rectum				
Clark[16]	16	M	Sigmoid	Abdominal pain	6 months	6 months three days	Adenocarcinoma
Pillon[85]	16	F	Rectum	Abdominal pain	3 months		
Grulee[40]	16	F	Sigmoid	Abdominal pain	.....	.....	Adenocarcinoma
azarus-Barlow[56]	16	M	Rectum	.....	.....	.....	Colloid
Porter[87]	16						
Martin, C. F.[65]	16	M	Rectum	Bleeding	8 months	15 months	
Rankin and Chumley[90]	16	..	.....	.....	.....	.....	Colloid
Steffins and Burke[108]	17	M	Rectum	.....	.....	.....	Colloid
Orr[77]	17	M	Rectum				
Gant[33]	17	M	Rectum				
Gant[33]	17	F	Rectum				
Lawson[55]	17	M	Rectum	Bleeding	7 months	7½ months	Colloid
Senn[102]	17	M	Rectum				
Colwell[18]	17	M	Rectum				
Oehler[75]	17						
Allingham[2]	17	M	Rectum	.....	.....	.....	Encephaloid
Foges[30]	17	M	Rectum	.....	8 months		
Schoenning[100]	17	F	Rectum	Pain in rectum, bleeding			
Schoenning[100]	17	F	Rectum	Abdominal pain and constipation			
Mathews[66]	17	F	Rectum	.....	2 years	2 years	
Porter[87]	17	..	Rectum				
Quenu[88]	17	F	Rectum				
Hayden and Shedden[42]	18	F	Rectum				
Gant[33]	18	F	Rectum				
Steffins and Burke[108]	18	M	Sigmoid				
Gant[33]	18	F	Rectum	.....	.....	.....	Colloid
Lowenberg[59]	18	M	Rectum	Diarrhea	6 weeks	.....	Adenocarcinoma
Spittler[107]	18	M	Rectum	Constipation bleeding	3 months	6 months after operation, 10 months after first symptom	Colloid
Billroth[9]	18	..	Rectum				

CHART I (*Continued*)

	Age	Sex	Location	Symptoms	Duration of Symptoms	Duration of Life	Type of Tumor
Heuck[45]	18	M	Rectum	Constipation, pain in abdomen	17 months		
Hayd[44]	18	M	Rectum	Abdominal pain, bleeding	18 months	.....	Scirrhous
Miles	18	..	Rectum	.....	.....	.....	Carcinoma
Hastings[43]	18	M	Rectum	.....	4 months	14 months	
Csesch[20]	18	M	Rectum	Bleeding	8 months	.....	Adenocarcinoma
Cripps[19]	18	M	Rectum	.....	4 months	10 months	
Carnett[14]	18	M	Rectum				
Steffins and Burke[108]	19						
Smith[106]	19	M	Rectum	.....	.....	2 years after colostomy	Adenocarcinoma
Ross[96]	19	M	Rectum	Abdominal pain	2 weeks	4 weeks after onset	
DaCosta[23]	19	M	Rectum	Constipation	4 months	.....	Adenocarcinoma
Gant[33]	19	F	Rectum				
Molliere[73]	19	F	Rectum				
Miles	19	..	Rectum	.....	.....	.....	Carcinoma
Billroth[9]	19	..	Rectum				
Dunbar[27]	19	M	Sigmoid	Abdominal pain	2 months	.....	Colloid
Gurlt[41]	19	..	Rectum				
Phifer[83]	19	M	Sigmoid	Loss of weight and diarrhea	6 months		
Barber[6]	19	M	Sigmoid	Intestinal cramps	1 year	.....	Adenocarcinoma
Rohde[93]	19	M	Rectum	Bleeding			
Platt[86]	19	M	Rectum	.....	8 months	Living at end of one year	
Jacobs[86]	19	F	Rectum				
Dieterich, H.[26]	19	F	Rectum	.....	.....	.....	Adenocarcinoma
Grave[39]	19	M	Rectum				
Kiger[51]	19	F	Rectum				

By comparison of these tables it will be noted that abdominal pain was the most frequent symptom in youth, whereas bleeding was the most common in adults.

TABLE III

	No.	Per Cent
Bleeding	574	31.5
Change in bowel habit		
Constipation	289	15.8
Diarrhea	269	14.7
Frequent or urgent desire for stool	195	10.7
Pain:		
Discomfort (local)	101	
As pain or tenesmus	391	
Total	1819	

The histologic type of tumor was reported in only thirty-seven instances. (Table IV.)

TABLE IV

Adenocarcinoma	19
Colloid	10
Scirrhous	2
Encephaloid	1
Gelatinous	1
Epithelioma	1
Lymphosarcoma	2
Sarcoma	1

As Rankin has said, the active tissues of youth, instead of resisting invasion of carcinoma, invite its spread, and the young person who is host to malignant neoplasm has little chance for longevity, regardless of the type of therapeutic measures instituted.

Shedden,[103] in a report of thirty-six collected cases, states that in twenty-five

operation was done, while eleven were untreated. Of these, all except three of those operated upon were dead at the end of twenty-four months, and twenty-five of the original thirty-six were dead within nine months. In our collected series the duration of symptoms at the time of diagnosis, as estimated in thirty-four cases, was 6.9 months. The average duration of life as mentioned in eight cases was ten months and three days.

SUMMARY

1. A case of early carcinoma of the rectum in a male 4 years and 7 months of age is reported.

2. The literature has been reviewed, citing 123 authentic cases of primary malignancy of the anus, rectum or sigmoid colon below the age of 20.

CONCLUSIONS

1. Any patient, independent of age, who complains of pain in the abdomen, rectum or lumbar region deserves a complete study of the lower bowel, employing digital examination, proctosigmoidoscopy, and roentgenography.

2. There is no true cancer age.

3. All tissues removed should be examined microscopically as a routine procedure.

4. Thorough periodic examinations should be made for recurrence, metastasis, and other neoplasms.

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HODGKIN'S TUMOR OF THE ANTERIOR MEDIASTINUM AND ANTERIOR CHEST WALL*

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THE treatment of tuberculosis affecting the lymphatic glands of the neck is frequently surgical. The number of patients seen today suffering from this once common disease is few. The differential diagnosis of enlarged cervical glands usually depended on the result of surgical biopsy and not infrequently cases of Hodgkin's disease were discovered. With diminution of the number of cases of tuberculosis of the cervical lymph chains has apparently also come an apparent decrease in the number of cases of Hodgkin's disease affecting the cervical glands. Whether this diminution is more apparent then real and whether there is any connection between the decrease in the number of cases of tuberculous glands of the neck and Hodgkin's disease affecting the same lymphatic channels I am unable to state.

Hodgkin's tumor of the anterior mediastinum and anterior chest wall is an uncommon condition. Muller recently reported a case of single mediastinal tumor due to Hodgkin's disease. According to Pfahler and others this tumor is radiosensitive and should be treated accordingly when the diagnosis has been positively affirmed. When there is doubt, as there was in the patient under discussion, operation is advisable, if for no other purpose than that of obtaining a biopsy. If, however, biopsy is the only objective, a formal operation is probably not necessary, since with the aid of fluoroscopic guidance and an 18 gauge needle, enough of the tumor can be aspirated for microscopic examination. In fact recently, after discussing the subject with Dr. James Ewing, I tried this method successfully on a patient with a tumor of the posterior mediastinum during the course of an exploratory thoracotomy.

Hodgkin's disease has been described in the past as producing either a benign or malignant tumor, having the structure of a granuloma, although the true nature of the disease according to Arons is still undetermined. Because of this uncertainty the tumor should be subjected to *x*-ray treatment, since, as stated before, tissues affected by Hodgkin's disease are radiosensitive.

Hodgkin's disease manifests itself in various forms; it may start as a universal enlargement of all the glands of the body, or be limited to the glands of the neck alone, or to a single tumor.

CASE REPORT

F. W., age 23, was admitted to the Jewish Hospital September 18, 1936.

A year and a half before admission a small lump was noticed on the upper left chest near the sternum. The tumor had been incised because it was thought to be an abscess of tuberculous origin, most likely a lymph node. *X*-ray treatment was given and the lump subsided somewhat but not completely. A short time before the patient's admission to the hospital the tumor began to enlarge and several *x*-ray studies confirmed the diagnosis of a tumor of the chest wall.

Physical examination showed a firm nonmovable mass at the junction of the left clavicle and sternum, blending with these bones and extending several inches downwards. There was a definite area of dulness over the bulging mass, which extended from the clavicle to the fifth rib. There were no palpable nodes in the neck or inguinal regions. The diagnosis at that time was chondroma of the chest wall. There had never been any report of an accident.

* Read before the Laennec Society, March 29, 1938.

The patient had had the usual diseases of childhood, and in addition was born a deaf mute.

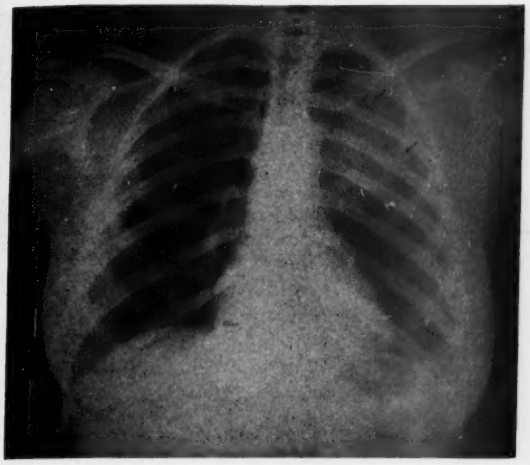

Fig. 1. *X*-ray before operation. Tumor definitely outlined, anterior portion of thoracic wall and extending under the sternum.

A communication from Dr. Pfahler stated that the tumor mass was approximately 10 cm. by 8 cm., occupying the left intraclavicular region; it was firmly attached to the wall of the chest and involved or developed from the anterior portion of the first and second ribs, or from the costal cartilages of these two ribs. There was no tuberculosis of the lungs or mediastinum. The tumor mass was not encapsulated. Partial destruction of the first and second ribs was present. The tumor was suggestive of chondroma or chondrosarcoma, with calcification. (Fig. 1.)

Laboratory Examinations. Blood count: hemoglobin 65 per cent; red cells, 3,650,000; white cells 9,100; polymorphonuclears 68; lymphocytes 31; eosinophiles 1; no abnormal leucocytes. There was slight achromia, anisocytosis, occasional poikilocytosis. The Wassermann was negative. Blood sugar was 115 mg., blood urea 16 mg., culture of the chest fluid sterile.

Operation. Under nitrous oxide and oxygen anesthesia, a semilunar incision was made around the breast including the left pectoral muscle. A cartilaginous inflammatory tissue enveloping the tumor was revealed after the ribs were removed. A hard inflammatory wall was stripped back from under the sternum and adjacent ribs; in doing so a thick creamy pus was obtained. When the sternoclavicular joint was disarticulated, pus exuded from the sternal ends of the first and second ribs. These two ribs were excised as far back as they were diseased. A small nick was made in the pleura, extending to the pleural cavity, which was repaired, without subsequent complications. After the tumor and the abscess cavity were dissected away the superficial fascia was closed with catgut. Michel clips approximated the skin.

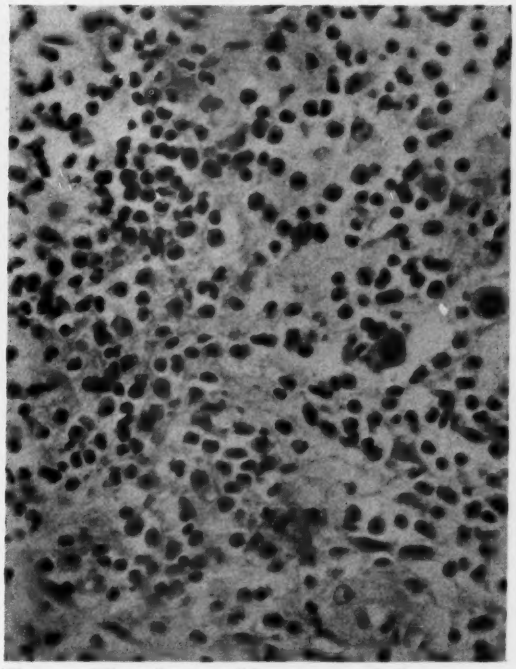

Fig. 2. The pleomorphic nature of the process seen in Hodgkin's disease is exemplified by the presence of lymphocytes, neutrophiles, eosinophiles and large mononuclear cells.

Pathologic Report (Dr. S. Levine). Specimen consisted of a large number of pieces of tissue, some soft and some bony in consistency. Section presented a large number of inflammatory exudative cells composed of polymorphonuclears, mononuclear cells, lymphocytes and a number of phagocytic cells. There were a large number of fibroblastic and epithelioid cells and also an endothelial proliferation, in addition to reticulum cells. No typical Dorothy Reed cells were noted. However, a number of large endothelial cells with hyperchromatic nuclei were present. Large areas of necrosis were seen throughout, and a considerable amount of fibrosis was observed. There was no definite evidence of tuberculosis. A number of eosinophiles were present.

The diagnosis was Hodgkin's granulomatous tissue undergoing acute inflammatory changes. (Fig. 2.)

In an *x*-ray report September 30, 1936, Dr. Leon Solis Cohen stated that both diaphragms were normally arched. The cardiac silhouette was normal in size, shape and position. The mediastinum showed no widening. The anterior portions of the left first and second ribs had been excised. The right lung was normal, while the left showed a diffuse haziness, most likely due to thickening of the parietal pleura or its involvement by the granulomatous mass. There was no definite evidence of a pneumothorax on the left side, but the lung was hypoventilated, probably due to fibrosis. There was no evidence of mediastinal Hodgkin's. (Fig. 3.)

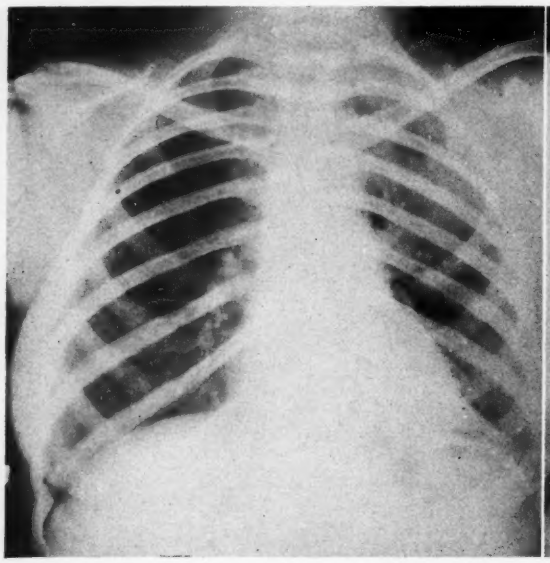

Fig. 3. Twelve days after operation. Diffuse haziness over anterior portion of thorax. Definite outline of tumor absent.

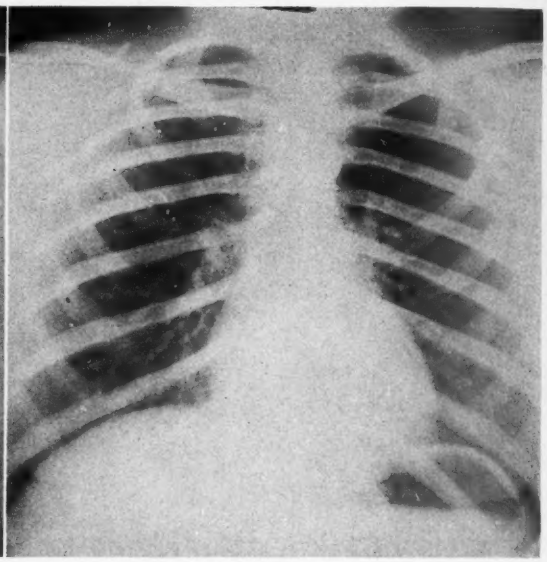

Fig. 4. Seventeen months after operation. Entire absence of mediastinal tumor. Portions of first and second ribs absent due to operation.

The patient received many *x*-ray treatments and on February 23, 1938 the heart shadow was reported as normal, the diaphragmatic shadows clear, and the lung fields and ribs as perfectly normal in appearance. No evidence of any mediastinal enlargement nor any Hodgkin's infiltration of any of the ribs was visible. The left first and second ribs were short, due to the former resection. (Fig. 4.)

SUMMARY

A case of Hodgkin's tumor of the anterior mediastinum and chest wall is presented. The condition is extremely rare. The diagnosis may be established by needle biopsy under fluoroscopic control or by surgical biopsy. *X*-ray therapy caused the tumor to become smaller, but it did not disappear. Therefore the remainder of the tumor was removed surgically. In the eighteen months since operation no recurrence has been noted.

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GAS GANGRENE OF THE ABDOMINAL WALL FOLLOWING RUPTURED GANGRENOUS APPENDICITIS

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GAS gangrene of the abdominal wall following appendicitis is a rare condition and very serious in nature. The acute, fulminating toxic type of gangrene, if not recognized early, rapidly proves fatal.

Almost all of these infections following abdominal operations are the result of the presence of the organism in the pathologic process or in the intestinal tract. The most common organism found is the Bacillus welchii.

Brown and Murphy,[1] in reporting a case of progressive postoperative gangrene of the abdominal wall following an appendectomy, state that Lynn of Baltimore has seen or collected from literature up to November, 1931 only fourteen such cases.

Gamble[2] reports eight cases of gas gangrene of the abdominal wall in 429 cases of appendicitis in which drainage was used. There were five deaths.

Shipley[3] reported a case of progressive ulceration of the abdominal wall which evidently followed drainage of a walled-off appendiceal abscess. The patient recovered after treatment with cautery and grafts.

Shearer[4] emphasizes the fact that gas gangrene of the abdominal wall responds to wide incision and drainage in enough cases to warrant the radical procedure, provided, of course, the patient's general condition will permit the treatment. His case followed gangrenous appendicitis; crepitus was felt forty hours postoperatively and a smear and blood culture showed pure cultures of Bacillus welchii. The patient died forty-four hours after operation.

Howe[5] reported a case following an operation for chronic appendicitis. The treatment consisted of wide opening of the abdominal wound, insertion of a drain to the peritoneum and administration of antitoxin. The Welch bacillus was not isolated. The patient received 180,000 units of gas gangrene antitoxin and recovered.

CASE REPORT

This case represents the sole instance of gas gangrene of the abdominal wall among 691 cases of acute and gangrenous appendicitis at the Bushwick Hospital (1934–1938).

The patient, a white male, aged 32, was admitted January 13, 1938 with complaints of severe generalized abdominal pain, nausea and vomiting. These had been present for three days, during which time the patient had taken neither solid nor liquid food. The pain had begun in the region of the epigastrium and had then passed to the lower right quadrant. Each attack of pain was accompanied by a feeling of warmth.

Physical examination showed that the patient was dehydrated, with dry, coated tongue. The abdomen was board-like, showed direct and rebound tenderness and there was a positive Blaisdell sign. No masses were felt. Acute ruptured appendicitis with peritonitis was diagnosed.

The white blood count was 29,950, with 90 per cent polymorphonuclear cells. The urine was positive for acetone.

After the administration of 1,000 c.c. of 5 per cent glucose in saline intravenously, the abdomen was opened. An acute gangrenous ruptured appendix with local peritonitis was found. The omentum was plastered around the appendix and both were lying over the urinary bladder. Three inches of the tip of the omentum were gangrenous. The peritoneum was thickened and indurated. No free fluid was present in the abdominal cavity. The appendix was removed with the actual cautery and the gangrenous tip of the omentum was removed by ligation and excision. The abdominal wound was closed in layers without drainage. Another

intravenous injection of 2,000 c.c. of 5 per cent glucose was given.

For two days postoperatively the patient's condition was fair and seemed to improve. His temperature dropped from 104 degrees to 101.6. Dehydration was still present and he appeared definitely toxic. Intravenous glucose was continued. On the third day, the patient vomited dark, greenish fluid and complained of abdominal pain. Twenty-four hours later two tension sutures and two skin clips were removed from the wound and a foul-smelling, dark fluid escaped from its lower edge. The skin was dark and crepitus could be felt on both sides of the wound. The temperature was 104.4°F., the pulse 140 and the respirations 30 per minute.

Aerobic and anaerobic cultures were taken from the wound and showed Cl. welchii. Five thousand units of gas bacillus and tetanus antitoxin were given intravenously immediately, followed by 23,700 units of polyvalent gas gangrene antitoxin. A Wangensteen drip apparatus was used because the patient continued to vomit.

On the following day, the fifth after operation, the patient was delirious and his condition definitely worse. The wound still discharged foul-smelling fluid and crepitation was advancing to the chest and groin. An additional 23,700 units of polyvalent gas gangrene antitoxin were given intravenously. However, the temperature remained high, the pulse rate went to 150 and then to 160, respirations advanced to 44 per minute, and the patient's condition was critical. The dose of polyvalent gas gangrene antitoxin was repeated in the afternoon, but shortly after midnight he died.

No post-mortem examination could be done. However, an *x*-ray report, made on the fourth day after operation, showed gas infection of the abdominal wall, obstruction in the upper ileum and peritoneal effusion. The pathologic report covering the specimens removed at operation showed an acute, gangrenous, ruptured appendix, an omentum dark red in color and covered with a plastic grayish exudate.

CONCLUSIONS

1. The acute fulminating toxic type of gas gangrene of the abdominal wall following appendicitis, as illustrated by this case, is a rare and rapidly fatal condition.

2. Polyvalent gas gangrene antitoxin (71,100 units) and 5,000 units of gas bacillus and tetanus antitoxin were of no avail.

3. Multiple wide incisions over the areas of crepitation, as advocated by Shearer,[4] were not made.

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NEW INSTRUMENTS

A PNEUMATIC DYNAMOMETER

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INJURIES to the upper extremity are often followed by weakness and disability of the hand. In determining the individual's ability to work it is necessary to evaluate the strength of the hand, as the grasping power is of chief importance in the function of the entire limb.

In order to measure this disability accurately, the writer has devised a pneumatic form of dynamometer which has been used with much satisfaction. This instrument combines an air gauge and blood pressure bulb connected with rubber tubing. Its soft rubber bulb is easily squeezed even in tender or partially stiff hands, and there is a sufficient range in the calibrations to register from a feeble to the strongest grasp. The gauge is calibrated both in pounds and in percentages.

For examinations the escape valve is tightened, thus holding the indicator at the maximum figure. Such an apparatus is also helpful for daily exercises, as the injured person can note his progress and thus is encouraged during recovery. For exercising, the valve is kept open, so that the needle oscillates.

The satisfactory results obtained with this dynamometer in the examination of industrial cases suggests its use also in neurology and athletics.

The writer wishes to express his appreciation to Charles Lentz and Sons for help in the construction of this instrument.

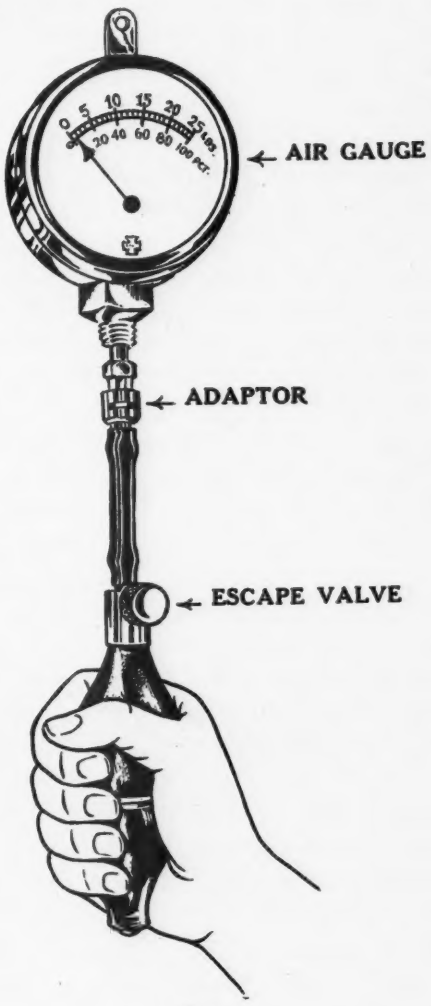

FIG. 1.

A NEW TYPE OF BLOOD TRANSFUSION APPARATUS*

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THE instrument[1] herein described operates on a new principle and is so simple and reliable that with its use the direct transfusion of whole blood becomes a trouble-free procedure.

The transfused blood passes from the donor's vein to the recipient's vein through a short piece of rubber tubing and never comes in contact with any part of the instrument.

The instrument is constructed entirely of metal, has no breakable parts and fulfills all the factors of safety required of a transfusion apparatus.

Principle. A piece of rubber tubing (*C*) lies in a channel on top of a large worm (*D*), as shown in Figure 1. The tubing is held against this worm by means of a bar (*B*) so that it is pinched at the points where it comes in contact with the worm (*A*). As the worm rotates its edges slide along the rubber tubing. This creates suction at one end of the tubing and pressure at the other. Continued rotation of the worm causes a continuous stream of blood to flow through the tubing.

The amount of blood delivered depends on the inside diameter of the tubing. The tubing has therefore been calibrated and a counter on the instrument records the amount of blood delivered in *CC*.

The instrument is small, compact and of all metal construction. (Fig. 2.) It may be sterilized by boiling. It has a heavily weighted base (*O*), and will stand upright wherever it is placed. The base is attached to the instrument proper by means of a bayonet fitting so that with one twist, the base can be removed and the instrument held in the hand if so desired by grasping the upright (*L*).

A handle (*M*) is provided by means of which the worm (*D*) is rotated. *The handle can be turned only one way. This allows the blood to flow in one direction only and there can be no back-flow.*

A counter (*G*) which records the amount of blood in *CC* is mounted so that it can easily be read.

At the top of the instrument is a flat metal cover (*B*) held in place by two spring clips (*F*). Removal of the cover reveals the channel in which the tubing lies when the instrument is in use. Two pieces of glass tubing, a straight piece (*J*) and a T-tube (*H*) serve as observation windows.

For Transfusion. A few simple steps make the instrument ready for a transfusion. The cover is removed, 20 c.c. of glycerin is poured into the chamber around the worm, the tubing is placed in the channel and the cover is replaced.

Rotation of the handle while the donor's side of the tubing is immersed in saline will fill the tubing with saline. The transfusion may then be given.

Although the instrument is boilable, only the tubing need be sterilized unless the transfusion is to be given in a completely sterile set-up. Since the blood passes only through the tubing, never coming in contact with the instrument, a previously sterilized tubing, if always on hand, makes the instrument immediately available in an emergency.

Transfusion Statistics. During its development the instrument has been used in over 300 transfusions at the Kings County Hospital and in about 175 at the Brooklyn Jewish Hospital. All were given with uniform ease. There has been no clotting even though as much as 750 c.c. of blood was given on several occasions.

[1] The instrument is manufactured by Fred Haslam & Co., Inc., Brooklyn, New York.

* From the Hematology Service, Kings County Hospital, Brooklyn, New York.

In the 175 transfusions given at the Brooklyn Jewish Hospital, only two mild post-transfusion chills and one mild urticarial reaction were reported.

blood through the apparatus fifteen times, but no red cell destruction was observed.

Other Uses. The instrument, although primarily a transfuson apparatus, lends itself to the following other medical uses and in each instance simplifies the procedure:

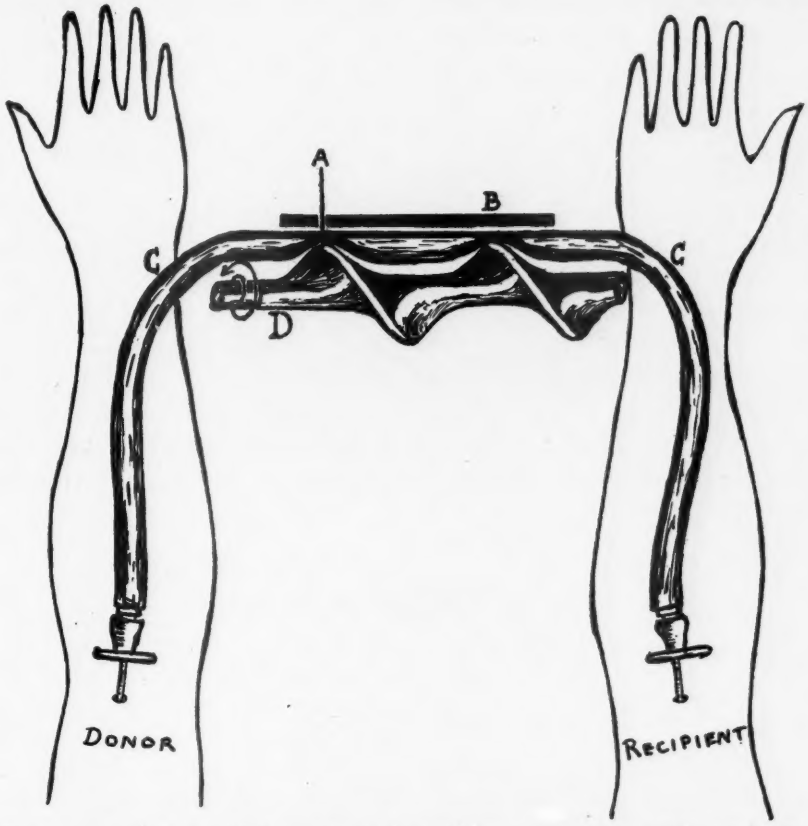

Fig. 1. A, point at which worm pinches tubing. B, cover, C, rubber tubing. D, worm. Schematic drawing illustrating principle upon which instrument operates.

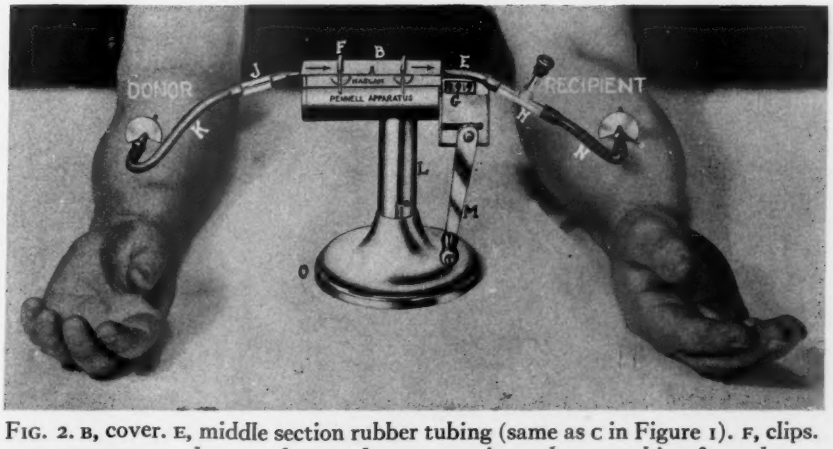

Fig. 2. B, cover. E, middle section rubber tubing (same as C in Figure 1). F, clips. G, counter. H, glass T-tube. J, glass connecting tube. K, tubing from donor. L, upright stand. M, handle. N, tubing to recipient. O, removable weighted base.

Dr. J. R. O'Sullivan of Kearny, New Jersey ran a quantity of citrated human

1. *Phlebotomy.* The instrument can be taken to the bedside and the usual messiness of phlebotomy eliminated. It also provides a simple means of collecting the blood in a sterile manner by inserting the delivery end of the tubing into a sterile flask containing citrate. The amount withdrawn is recorded by the counter.

2. *Aspiration.* By connecting the suction side of the tubing to the aspirating needle, continuous suction can be applied to chest or abdomen. The amount of fluid that can be aspirated is limited only by the desire of the operator or content of the cavity.

3. *Pneumothorax.* The instrument pumps air as well as blood. By attaching the pneumothorax needle to the delivery side of the tubing, and the upright limb of the glass T-tube (*H*) to the water manometer, one has an efficient pneumothorax apparatus. The necessity for water bottles is eliminated. The air may be pumped in as slowly as desired under manometric control and the amount introduced is recorded by the counter.

SUMMARY

A blood transfusion apparatus is described which operates on a new principle. It has no syringes or valves. It has the advantages of being very simple in operation, reliable, unbreakable and extremely efficient in the handling of whole blood. It is versatile and lends itself to other uses besides transfusion such as phlebotomy, aspiration and pneumothorax.

SPECIAL ARTICLE

Formation of Pelvic Viscera and Their Outlets on the Surface

BY

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FORMATION OF PELVIC VISCERA AND THEIR OUTLETS ON THE SURFACE*

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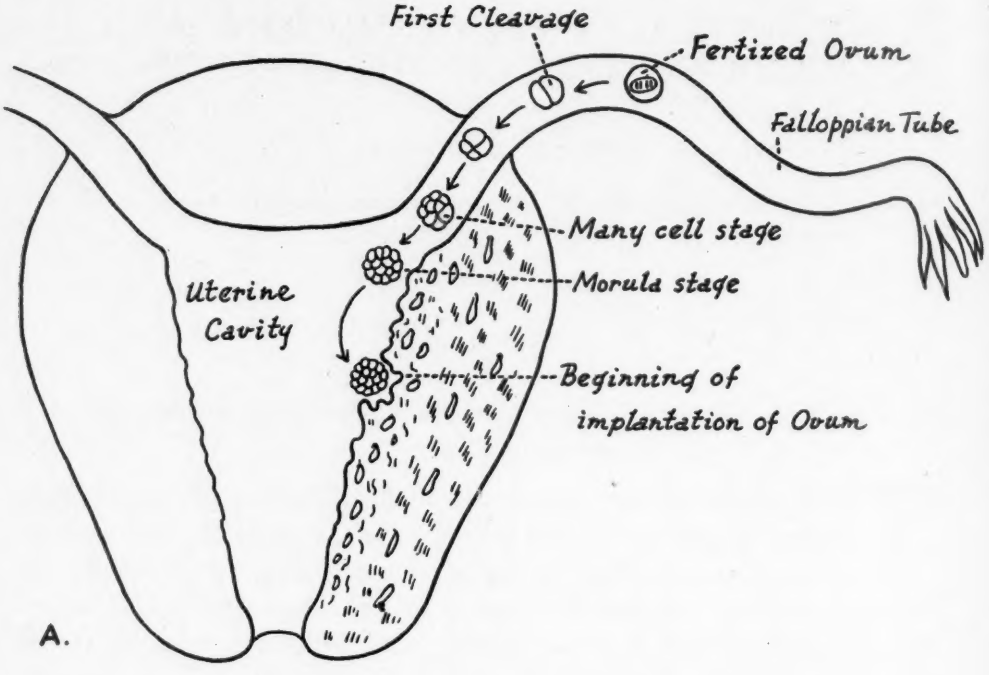
PART I

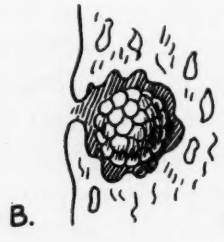
MATURATION—FERTILIZATION—ZYGOTE—EMBRYO TO FORMATION OF INTERNAL CLOACA

THE anatomic history of an individual vertebrate begins with the life history of its parental germ cells. These cells are early laid aside as gonocytes in a small strip of mesothelium on the ventromedial border of the urogenital folds. Here the gonocytes proliferate extensively and become lodged in their differentiated sex glands as the female sex cells or oögonia of the ovaries; the male sex cells or spermatogonia of the testes. The gonocytes during their active stage of multiplication resemble the body cells—soma cells—in their structural appearance and in their mitotic process of equational chromosome division in which the number of chromosomes remains constant for each cell. The primordial sex cells eventually cease multiplying and enter upon a stage of growth in preparation for two specialized and final divisions. The descendants of the gonocytes that have attained the period of growth—primary oöcytes in the ovaries, and primary spermatocytes in the testes—realign their paired chromosomes, one derived from each parent, in single segments whose number is half that of the chromosome number.

The primary spermatocyte divides into two equal and smaller secondary spermatocytes, each of which acquires one of the paired chromosomes forming each segment. By this reductional division—miosis—one half of the chromosomes has been eliminated from each of the two secondary spermatocytes, and the sex chromosome resides in but one of the two secondary spermatocytes. The two

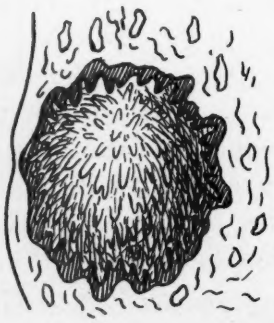
* Acknowledgment is made to the Department of Anatomy of New York University Medical College for the privilege of using its facilities. The author also wishes to express his deepest gratitude to Miss Mary Lorenc for her painstaking work in the illustration of this article. Dr. M. J Horan, formerly of the Anatomy Department of Fordham Medical College, has given advice and encouragement during the progress of this work. I am grateful also to Mr. Billy Rose for his kind aid.



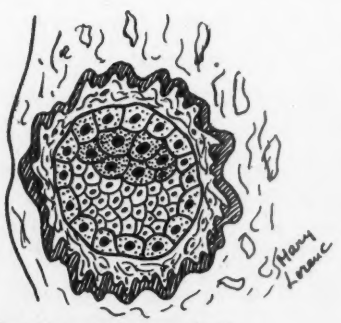


Implantation of ovum.

Sealing of point of entry.



Formation of Trophoblast.



Section through D.

PLATE I. A, uterus and fallopian tube with fertilized ovum, showing cleavage and beginning implantation. B, implantation. C, sealing of point of entry of ovum. D, formation of trophoblast on wall of zygote. E, section through D to show inner cell mass.

secondary spermatocytes undergo the final second division by equational splitting of their chromosomes and yield four equal smaller spermatids of which two possess the sex chromosome and each contains half the number of chromosomes fixed for the species. The spermatids are each transformed into a spermatozoon by a complex process which provides the matured male sex cell—male gamete—with a locomotor apparatus. The spermatozoon has the appearance of a lance 0.05 mm. long.

The primary oöcyte divides into two markedly unequal cells by the same reduction process that took place in the primary spermatocytes. The very small cell—first polar body—contains half the chromosomes and little cytoplasm; the large cell—secondary oöcyte—contains half the chromosomes and almost all of the parent cytoplasm. Each of the two cells undergoes the final second division by mitosis and four cells are formed. The first polar body divides into two cells; the secondary oöcyte divides into a small cell—the second polar body—and a large cell—female gamete—the matured ovum. Each of the four cells contains one half the number of chromosomes fixed for the species. Of these four cells only the matured ovum, after fertilization, is capable of developing a normal individual. Fertilization of the polar bodies may give rise to monster formations. The female gamete is a spheroid body whose greatest diameter is 0.25 mm.

The ovarian follicle containing a secondary oöcyte, distends, and upon rupturing liberates its oöcyte which is then guided by the ciliary current of the oviduct down its lumen. In the outer portion of the tube, a spermatozoon pierces the outer layer of the secondary oöcyte and enters its cytoplasm. The secondary oöcyte gives off its second polar body and becomes the mature ovum whose nuclear material forms the female pronucleus. The head of the spermatozoon, practically all nuclear material, is transformed into the male pronucleus which is accompanied by the two centrosomes that reside in the middle piece, and the flagellum is absorbed. The male pronucleus is smaller than the female pronucleus. Each pronucleus contains one half the number of chromosomes typical for the species.

The male and female pronuclei fuse and form a single nucleus—segmentation nucleus—and fertilization is completed. The cell resulting from the union of the male and female gametes is the fertilized ovum—zygote—to which the full number of chromosomes fixed for the species has been restored. The zygote has the capacity of developing into a new individual of the species, whose sex is determined by the chromosome constitution of its nucleus. Through

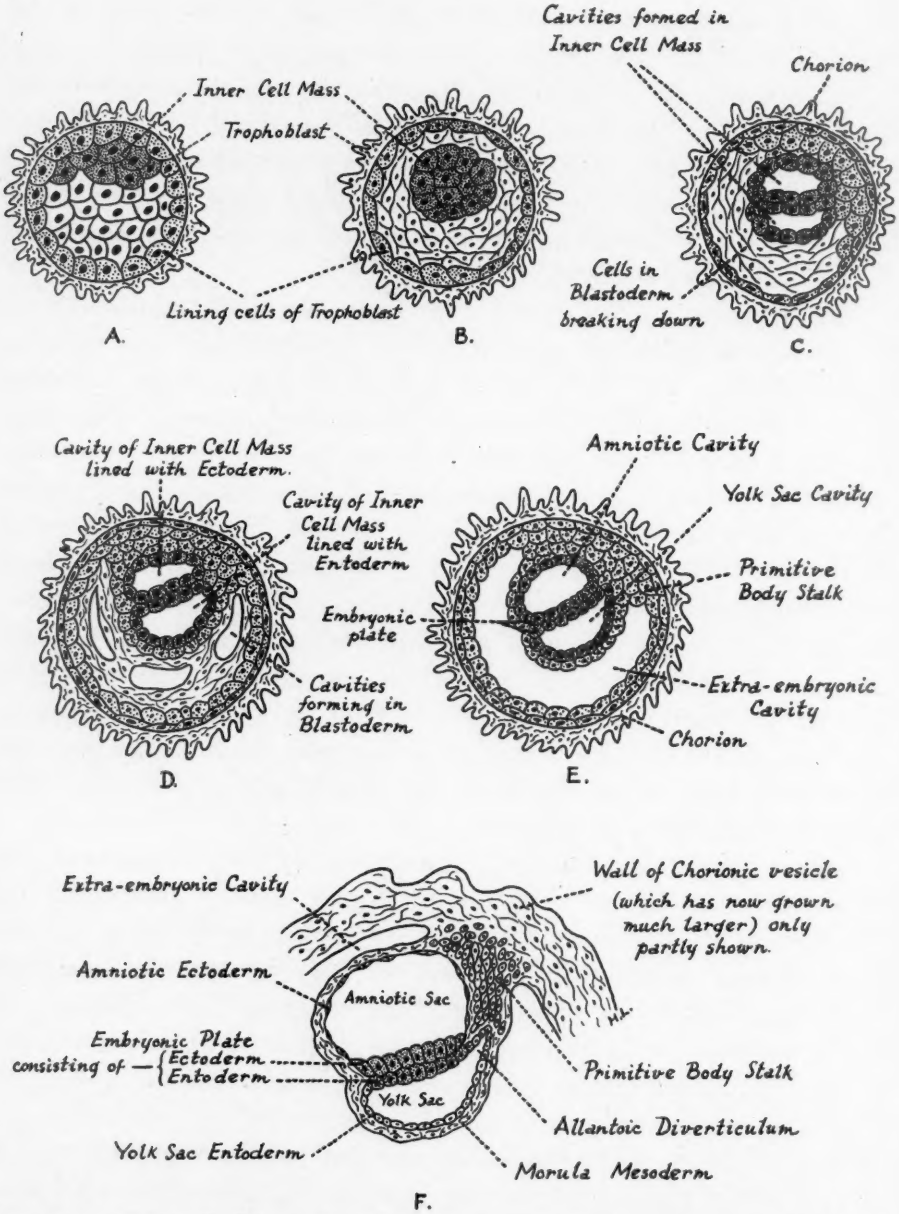


PLATE II. A, same as E in Plate I. Zygote, showing inner cell mass and surrounding tissue. B, zygote lining cells of trophoblast. Inner cell mass separated from wall of blastoderm. C, cavities formed in inner cell mass, surrounding cells in blastoderm breaking down. D, cavities forming in morula mesoderm of blastoderm. E, formation of primitive body stalk. Amniotic and yolk sacs, and completed extra-embryonic cavity. F, identification of primitive structures; the embryonic plate with its cells differentiated from cells lining the amniotic and yolk sac cavities.

the genes of the chromosomes it is endowed with all the potentialities of its parents.

The zygote immediately enters upon the period of segmentation. By a series of consecutive mitotic divisions it develops into a large number of cells which are grouped together in the form of a solid spheroidal mass, the morula. Early in the segmentation process the cell cleavages appear equal in size, and later the divisions result in the formation of cells of different sizes and apparently different functions. A few cells retain all the potentialities derived from their parents and develop into the parental or primordial germ cells. To the remaining cells only parts of the chromosome inheritances are transferred and they develop into the soma cells whose descendants are the tissue cells of the body.

The zygote, scarcely changed in size during the stage of segmentation, easily reaches the cavity of the uterus, where a special cellular formation of the mucous membrane—decidual reaction—has taken place. The outer layer of the cell mass of the morula develops into a syncytial covering layer—trophoblast—which exerts a digestive action on the decidua. The uterine epithelium in contact with the zygote is destroyed and the zygote sinks into the uterine mucosa where it becomes enclosed by the mucous membrane growing over it.

Until implantation takes place, the zygote is a free organism in the uterine cavity dependent upon its own small reserves for nourishment. Consequently during this period it hardly grows. Upon implantation, the zygote becomes a parasite upon the maternal organism from which its nourishment is derived throughout the remainder of the intra-uterine period. With the establishment of the parasitic relationship the ovum enters upon a period of extremely rapid growth.

The morula cells of the zygote arrange themselves in a cluster—inner cell mass—suspended from the outer enveloping layer of trophoderm in a bed of slightly differentiated cells—morula mesoderm—developed from both inner cell mass and trophoderm. The morula mesoderm grows completely around the inner cell mass and separates the cell mass from the trophoderm. The detached inner cell mass lies in the bed of morula mesoderm.

Two clefts appear in the detached cluster of cells and expand into the two closely apposed vesicles of the future embryo, the external or amniotic vesicle and the internal or yolk vesicle. In the morula mesoderm, spaces appear and on becoming confluent form a single large cavity—extra-embryonic acvity of the embryo.

The bivesicular structure ensheathed in morula mesoderm lies in a bulge of the wall of the extra-embryonic cavity.

The zygote has been transformed from a solid spheroidal struc-

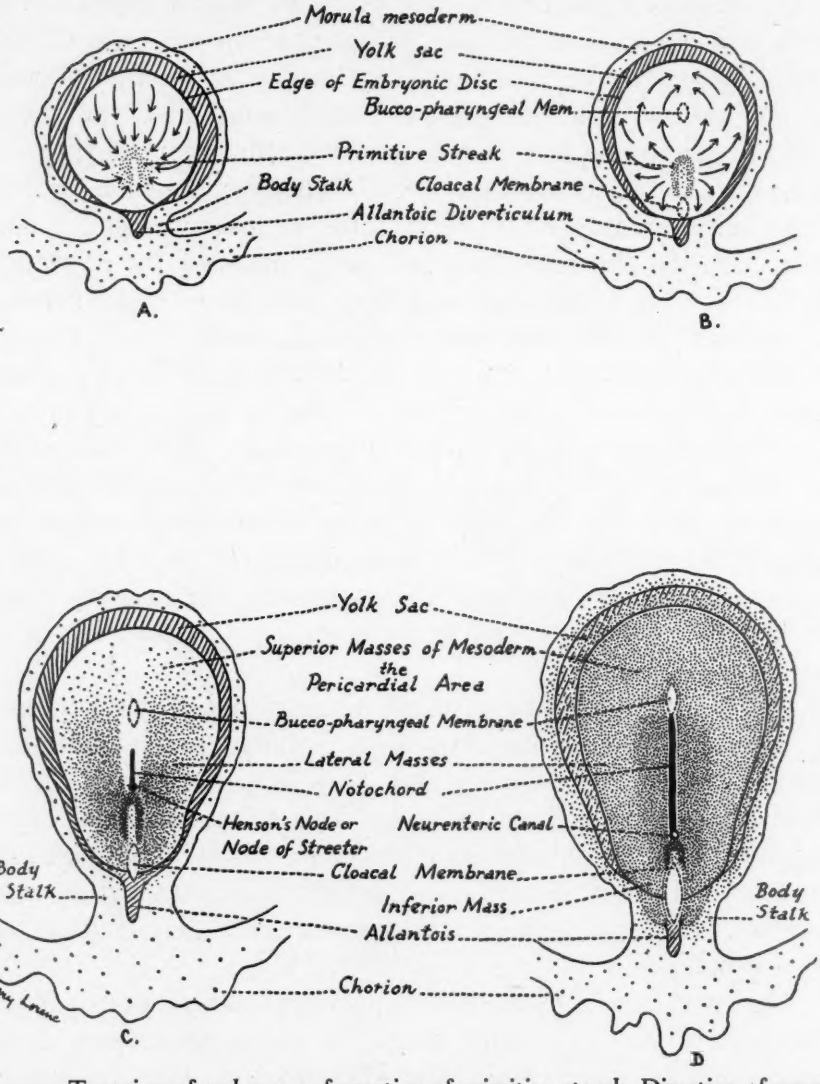


PLATE III. Top view of embryo. A, formation of primitive streak. Direction of wave of activity. B, direction of wave of proliferation of mesoderm from primitive streak. C, mesodermic wings extending through plate, beginning of notochord. D, mesodermic wings complete, notochord complete. Neurenteric canal.

ture into a large vesicle containing two smaller ones in its medial wall. Each of the two small vesicles is lined on its internal surface by a continuous layer of cells. The linings of the two vesicles are applied to each other in the small area where the vesicles are apposed, and form the bilaminar disc, shield, or plate of the embryonal

area.* The external or amniotic vesicle is lined with ectoderm and its floor is the dorsal lamella of the embryonal disc. The internal or yolk vesicle is lined with entoderm and its roof is the ventral lamella of the embryonal disc. The ventral lamella of entoderm continues as the lining of the primitive alimentary tract and its appendages. The dorsal lamella of ectoderm provides the material for the development of the remainder of the embryo.

The bivesicular structure lies in a sheath of morula mesoderm—the primitive body stalk—that extends from the inner wall of the zygote. Orientation of the embryonal disc is determined by the portion that is attached to the body stalk which is the caudal, posterior, or lower region; the portion nearest the extra-embryonic cavity is the cephalic, anterior, or upper portion. The borders between the anterior and the posterior margin are the lateral margins.

The growing disc of the embryonic area becomes pear-shaped with the appearance of a linear groove—primitive groove—with low marginal ridges in the posterior fourth of the external surface. The area marked off by the primitive groove and its marginal ridges constitutes the primitive streak and is the zone of intensely active division of the cells of the ectoderm from which the embryonic mesoderm is formed by proliferation and migration. The embryonic mesoderm, the ectoderm and the entoderm comprise the three germinal layers of the embryo. Posterior to the primitive streak and further caudad the plate including its junction with the body stalk is composed of ectoderm closely applied to entoderm to form the cloacal membrane.

The cloacal membrane is distinct from the primitive streak and extends for a short distance on the body stalk. The growing sacks of the vesicles by their internally advancing portions stretch the intervening wall of morula mesoderm into a thin sheath lining the extra-embryonic cavity; the externally advancing portion of the amniotic sac reduces the broad base of the primitive body stalk to the narrower body stalk. The external portion of the growing yolk sac invades the body stalk by a slender diverticulum—allantois whose wall is partly bound by the cloacal membrane on the body stalk.

* The embryonal area of the zygote will form all of the future individual. The remainder of the zygote enters into the formation of the membranes and appendages which provide protection, nourishment, and support during intra-uterine life, and are cast off when the umbilical cord is severed at birth.

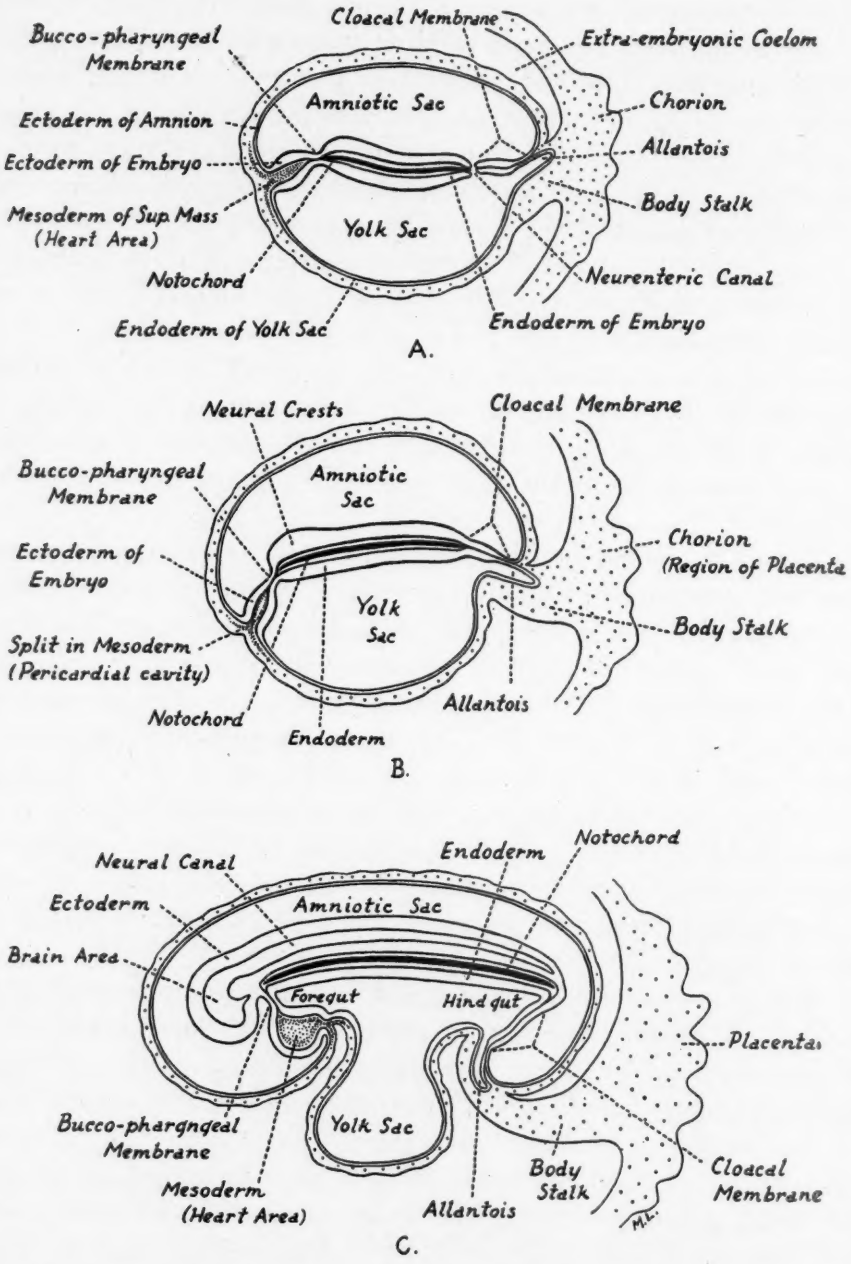


PLATE IV. Median sagittal sections. A, embryonic plate (with neurenteric canal open) being bent gently beyond ends of notochord by pressure of amniotic fluid. B, plate bent more, neurenteric canal closed. C, plate bent still more, forming head and tail folds for foregut and hindgut, drawing in of yolk sac. Extension of cloacal membrane on stalk.

The mesodermal cells from the lateral margins of the primitive streak where their proliferation is most intense, stream into the plate between the ectoderm and entoderm and condense into paired sheets. The lateral sheets grow into the plate in a paired wing-shaped formation and extend through the margin of the plate to fuse by their peripheral borders with the morula mesoderm that invests the bivesicular structure. The anterior crescents of the embryonal mesodermal formations meet cephalad to the buccopharyngeal membrane and fuse in a wide band to form the protocardiac area. The posterior crescents extend downward along each side of the cloacal membrane to the body stalk where they fuse with the morula mesoderm of the stalk. With the development of the embryonal mesoderm, the zygote becomes the embryo.

In a circumscribed area adjoining the cephalic end of the primitive streak—node of Streeter or Henson's knot—the proliferated cells from the ectoderm fuse with the underlying entodermal cells and enter upon an active state of multiplication. The cells form a column that penetrates the plate between the ectoderm and entoderm and fuses with the latter. The column occupies the space between the unfused crescent of the mesoderm formation up to the buccopharyngeal membrane. The mesodermal cells of the paired sheets are closely applied and distinct from this axial formation—notochordal process or head process. The developing notochordal process fuses with the underlying entodermal cells and draws away from the overlying ectoderm which sinks down and forms a groove—neural groove. The bulky mesoderm at each side of the notochord raises the margins of the groove in the overlying ectoderm which form the neural ridges or folds. The growth of the neural structures coincides with the growth of the notochord. The neural folds close the neural groove anteriorly by joining in a low terminal ridge which separates the groove from the buccopharyngeal area. Posteriorly the elevated folds diverge and extend into the prominent masses at each side of the primitive streak and embrace in the angle of their diverging margins the anterior portion of the streak and the notochordal node which become included in the neural floor. The notochordal process grows forward in a rod of multiple layered cells which are fused with the underlying entoderm. Cephalad, the layers of the rod become gradually reduced and fade into the entoderm immediately posterior to the fused ectoderm and entoderm of the buccopharyngeal membrane. A lumen appears in the notochordal process and rapidly forms a channel—canal of the notochord—which opens posteriorly, caudal to the notochordal node on the

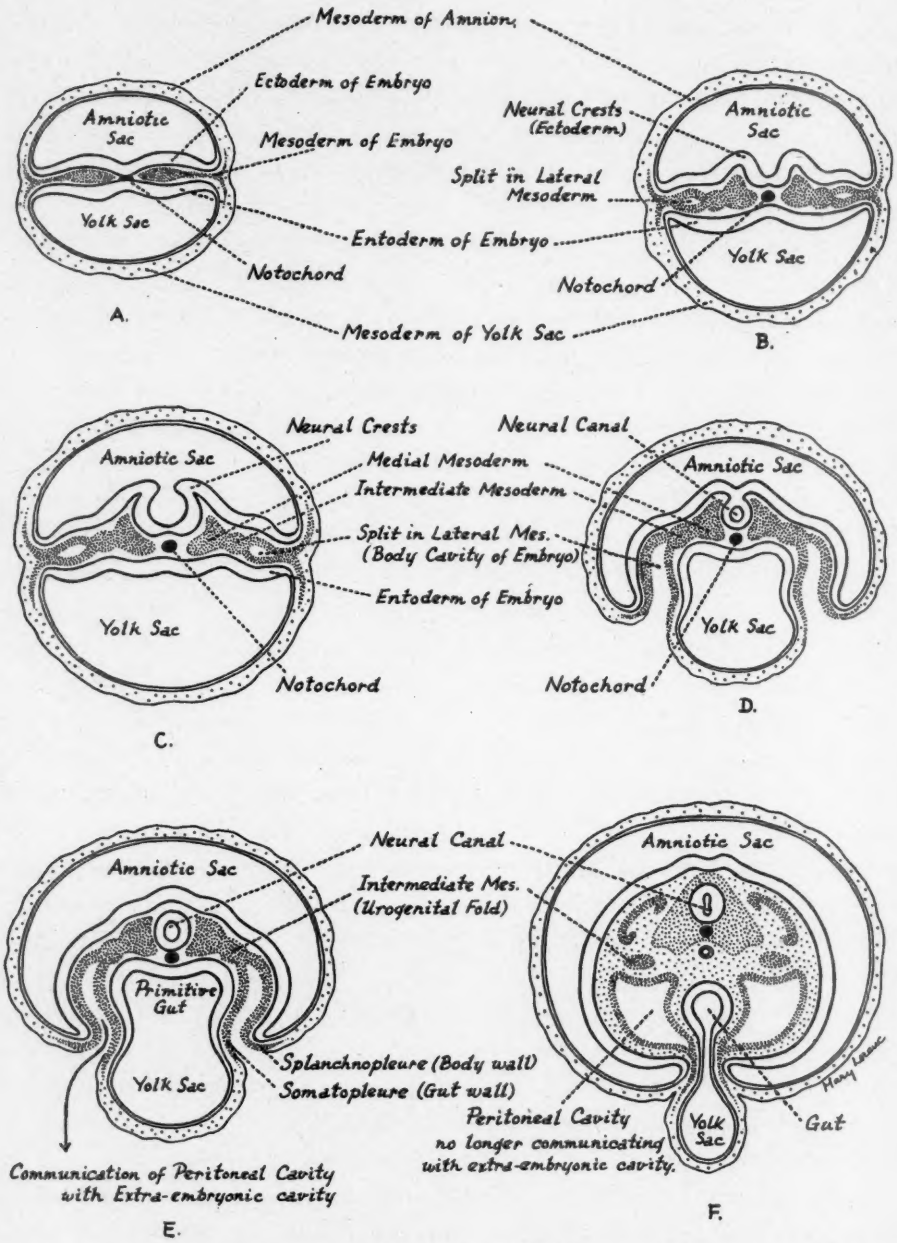


PLATE V. Transverse sections. A, embryonic plate. Ectoderm, entoderm and mesoderm. B, arrangement of three parts of mesoderm; beginning of split in lateral sheets. Rising of neural crests. C, larger split in lateral mesoderm, neural crests higher. D, embryonic plate bending over yolk sac, split in lateral mesoderm complete, forming open body cavity of embryo. Neural crests closed, forming neural canal. E, plate bent still more, forming primitive gut; yolk sac enclosed in amniotic folds, identification of splanchnopleure and somatopleure; body cavity still open to extra-embryonic cavity. F, plate folded so that lateral edges meet in ventral midline, squeezing off yolk sac to narrow communicating canal. Body cavity closed off from extra-embryonic cavity.

floor of the neural groove, into the amniotic vesicle. The canal of the notochord is separated from the cavity of the yolk sac by the floor of the canal that has fused with the entodermal roof of the yolk sac. Areas of this fused canal floor and entodermal roof break down and the canal which opens into the amniotic cavity by a single external orifice in the floor of the neural groove acquires openings in the cavity of the yolk sac. The yolk sac cavity is brought into communication with the amniotic sac by the persisting portion of notochordal canal—neurenteric canal—whose external orifice is on the floor of the medullary groove in the angle of the diverging neural ridges. The notochordal remnant—chordal plate—appears as a groove—notochordal groove—on the roof of the yolk sac. The entodermal cells from each side of the groove proliferate and meet each other to reform the cylindrical tube. The notochordal canal and its extension, the neurenteric canal, are quickly obliterated by the overgrowth of neighboring cells and the notochord is converted into a solid rod of cells. The cells of the neurenteric canal might be regarded as the rudimentary tissue of the future tail of the embryo.

The primitive streak becomes effaced by the caudad growth of the notochord. The posterior termination of the notochord extends to the upper end of the cloacal membrane between the

DERIVATIVE OF THE MESODERM

Paraxial	Intermediate	Lateral Mass
Sclerotome provides hard skeletal structures of the vertebral column and its ligaments, dura mater of brain and spinal cord. Myotome forms voluntary muscle.	Upper portion is faintly marked by transverse divisions and lower portion unsegmented as nephrogenic cord. Forms urinary apparatus except urethra and most of bladder. Genital glands and their ducts.	Precipitates out mesenchyme. Lining mesothelial cells of great serous cavities; pleural, pericardial, peritoneal. Connective tissue except vertebral column and head. Mesoderm of limbs. Probably all unstriped muscle fibers of wall of alimentary canal and blood vessels. Many cells, irregular in form and possessing ameboid activity appear between the definite layers of mesoderm and adjacent ectoderm and entoderm. These are the cells of the mesenchyme and they are derived from the mesoderm and possibly the ectoderm and entoderm. These cells have a definite reaction to certain dyestuffs.

medial origins of the crescentic margins of the inferior wings of mesoderm that border the cloacal membrane.

Each sheet of mesoderm lateral to its medial border is indented by a sagittal groove which extends through its length. The medial portion of the mesoderm is divided by a series of horizontal deep grooves into successive segments or somites. Each sheet is defined into three portions; a medial or paraxial portion that lies between the groove and the notochord; an intermediate region of the sagittal groove and the lateral cell mass that extends from the groove laterally to the margin of the plate where the embryonal mesoderm becomes continuous with the morula mesoderm of the wall of the extra-embryonic cavity.

Embedded in a knob of morula mesoderm the bivesicular structure of the zygote comprising two vesicles and the oval plate of their apposed surfaces has developed into the more highly differentiated bivesicular structure of the embryo. The amniotic sac growing against the dorsum of the broad base of the primitive body stalk has narrowed the stalk which becomes elongated. The yolk sac has extended into the body stalk to form the diverticular process of the allantois.

The bilaminar oval plate of the zygote has become incompletely trilaminar with the distribution of the embryonic mesoderm that is absent in the regions of the buccopharyngeal and cloacal membranes. The oval plate has become elongated by the development of the axial structures of the solid notochord and overlying heavy neural tube in the mid-longitudinal line. The plate has the form of a violin and suggests the adult form of the trunk with the pelvis. The differentiated plate, varying in resistance with the density of the structures, opposes a varying resistance to the pressure of the accumulating amniotic fluid and to the traction of the swelling amniotic sac. The plate is most resistant in the region of the longitudinal axis occupied by the solid notochord and heavy overlying neural tube, less resistant in the transverse axis by the mesodermal masses that extend from each side of the axial structures, and least resistant at the junctions of the anterior and posterior ends of the solid notochord with the fused ectoderm and entoderm of the buccopharyngeal and cloacal membranes where there is no mesoderm to strengthen the resistance to the developing pressure and traction.

The amniotic sac is attached to the dorsum of the body stalk and to the ectodermal rim of the embryonic plate which is the floor of the amniotic cavity. The accumulating fluid of the amniotic

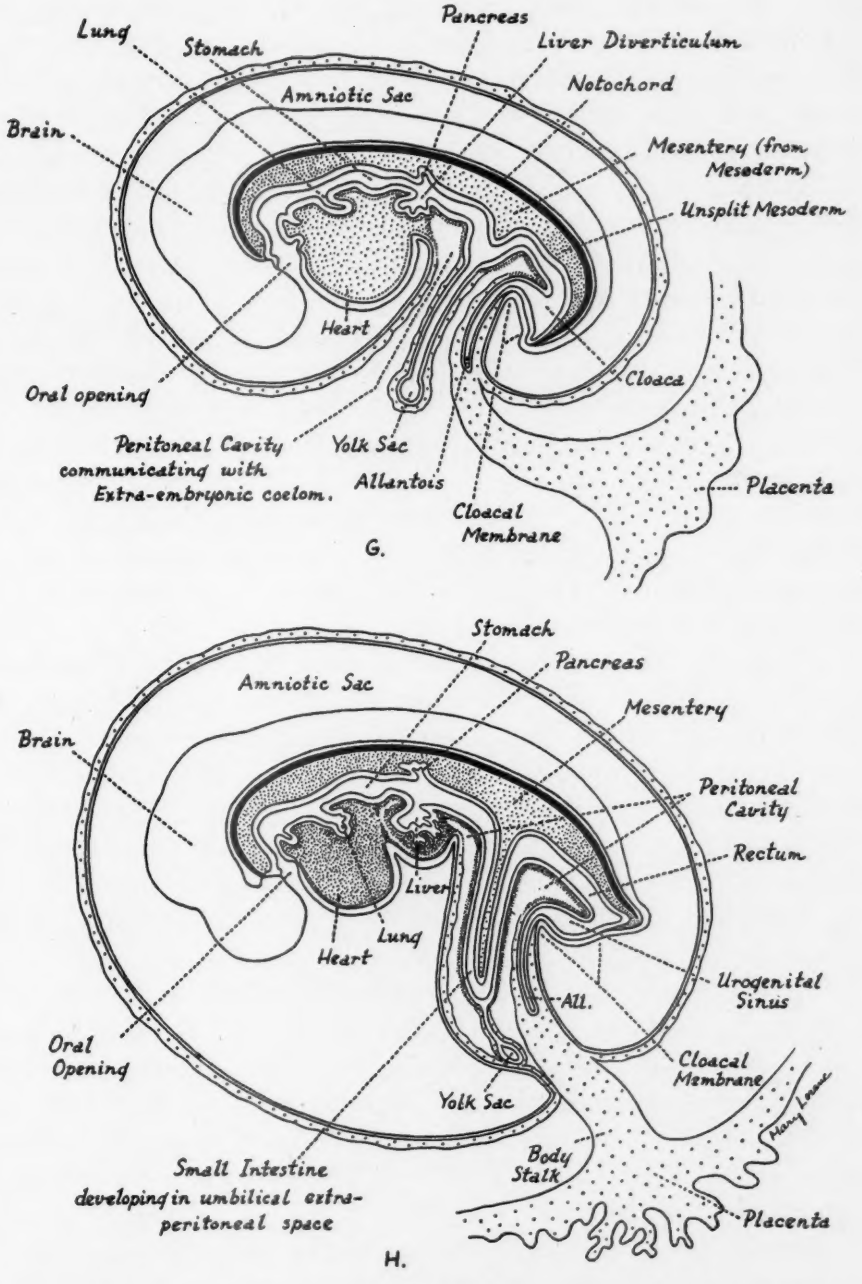


PLATE VI. Continuation of plate V. Median sagittal sections. G, formation of alimentary canal with its various diverticuli, formation of cloaca, lengthening body stalk—body cavity still open to extra-embryonic cavity. H, further development of alimentary canal. Small intestine forming in umbilical, extra-peritoneal space. Cloaca partly divided into urogenital cloaca and rectal cloaca.

cavity distends the rapidly growing sac which bulges beyond its attachments to the plate and stalk. The amniotic sac expands in a plane ventrad to the plate and advances medially with its attachments. The plate yields to the increasing weight of the amniotic liquid on its back and to the traction of the enveloping amniotic sac attached to its margins by folding ventrally at each end of the solid notochord to form the head and tail folds. Laterally, the pressure and traction form a gentle arching of the plate whose edges roll inward, taking with them the attached body stalk and yolk sac. The elongating stalk with its allantois is brought ventrocephalad to the posterior end of the notochord. The allantois forms an acute angle with the yolk sac and its lumen takes origin from the recess of the tail fold. The slowly growing yolk sac immediately beyond its attachment to the entodermal rim of the plate is compressed between the medially advancing folds of the amniotic sac to form the waist of the yolk sac—vitello-intestinal duct. The bending and arching of the embryo has transformed the flat plate into a cylindroid whose chamber is the primitive gut cavity.

The cavity of the medial portion of the primitive gut, the midgut, communicates widely in front with the yolk sac cavity through the large vitello-intestinal duct, cephalad through the anterior portal with the recess of the head fold—the foregut—and caudad by the posterior portal with the recess of the tail fold—primitive cloaca—which terminates ventrocephalad in the narrow channel of the allantois. The allantois, beginning as a diverticulum from the yolk sac, has changed its position to become the diverticulum of the primitive gut cavity.

The external surface of the tail fold between the caudal end of the notochord and the body stalk is occupied by the cloacal membrane which extends for a short distance on the under surface of the body stalk. The cloacal membrane, with its borders of mesoderm and covering of ectoderm, marks the external region or perineum of the future pelvic outlet. Caudal growth of the embryo beyond the cloacal membrane forms the tail.

Spaces appear in the mesoderm of the protocardiac area and within each lateral mass. The spaces become continuous. The spaces of each lateral mass become confluent and extend caudally as far as the inferior mesodermal wings of the tail fold to become the paired intra-embryonic spaces. The inferior mesodermal wings remain unsplit. Each intra-embryonic space extends peripherally through the mesodermal rim of the plate and the contiguous wall of morula

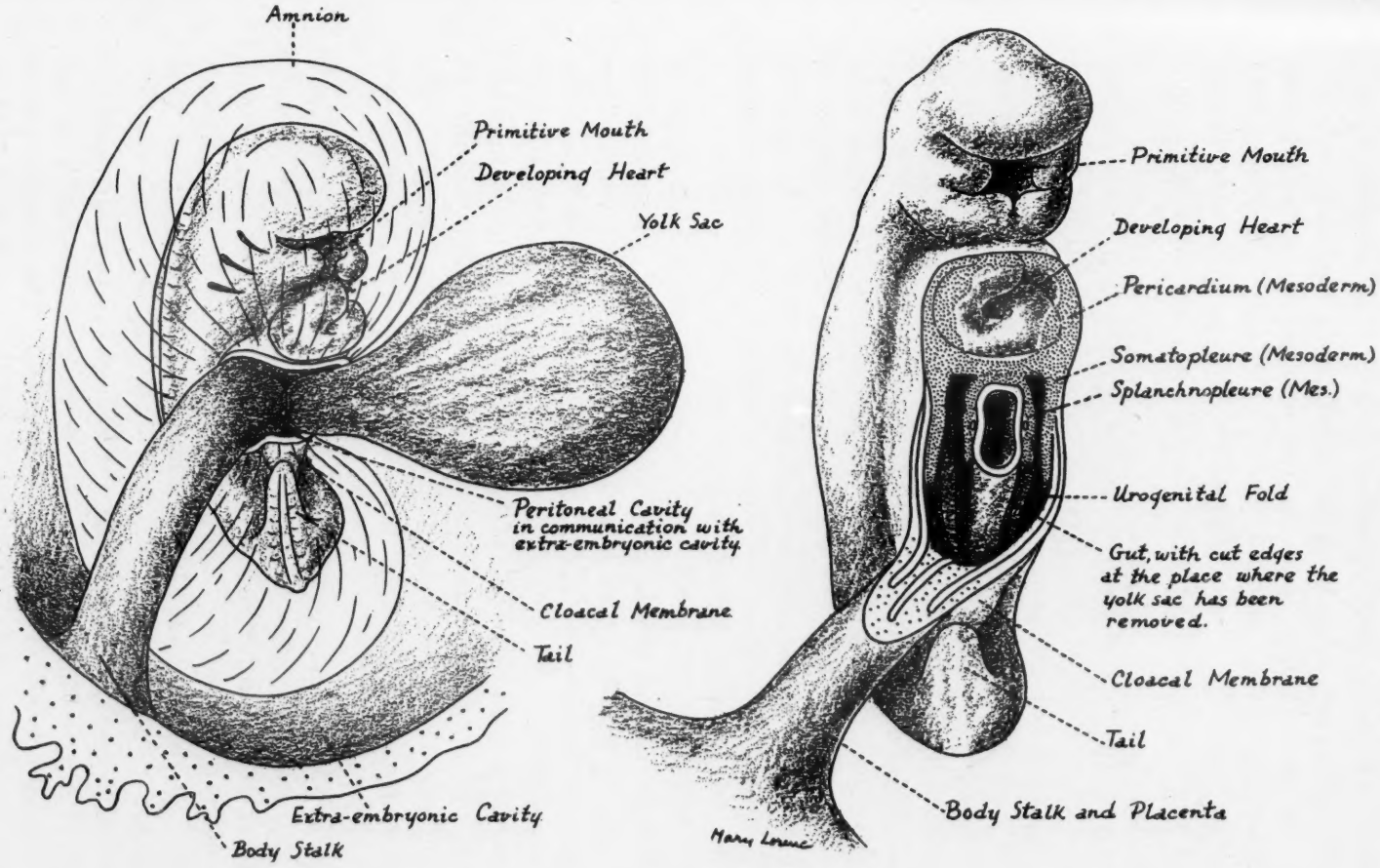


PLATE VII. External form of embryo. A, external form of embryo, enclosed in amniotic folds, body stalk and yolk sac emerging from belly of embryo. Body cavity still open to extra-embryonic cavity. B, external form, part of body wall and yolk sac cut off to show developing heart, enveloped in mesoderm, mesoderm of splanchnopleure and somatopleure, urogenital folds—peritoneal cavity.

mesoderm of the extra-embryonic cavity. The intra-embryonic space becomes continuous with the extra-embryonic space.

In the protocardiac zone the space remains confined to the mesoderm—pericardial cavity—from which paired dorsocaudal extensions—pleuroperitoneal channels—one on each side of the foregut open into the cephalic portion of each intra-embryonic space.

Each lateral mass of embryonic mesoderm is split by its cavitation process and forms two layers of mesoderm partially enclosing a cavity—cœlom—between them. The cœlomic cavity is continuous with the extra-embryonic cavity. The external layer of the split mesoderm remains applied to the ectoderm and forms with it the somatopleure. The corresponding internal layer of mesoderm remains applied to a portion of the entoderm of the primitive gut and forms with the entoderm the splanchnopleure. The splanchnopleures from both sides unite in front to complete the intestinal tube. The united splanchonopleures fuse with the wall of the vitellointestinal duct. Inferiorly, the union of the splanchnopleures closes over the primitive entodermal cloaca.

The alimentary canal extends from the buccopharyngeal membrane to the cloacal membrane; the attachment of the canal varies in its several portions. The foregut is attached to the walls of the entodermal pleuroperitoneal channels. The terminal foregut, midgut, and proximal hindgut are suspended in the body cavity by a continuous vertical mid-dorsal attachment to the angles of union of the split mesoderm of each side. The blood and nerve supply of the gut enters its posterior wall between the dorsal attachments. The dorsal attachments of the gut become attenuated and form the two leaves of its mesentery.

The hindgut caudal to its termination of the mesentery expands in a bay which occupies the interior of the tail fold and empties at its ventrocephalic border in the narrow channel of the allantois. The portion of the bay caudal to the origin of the allantois is the internal cloaca. The cloaca is lined throughout with entoderm.

The lateral walls of the entodermal cloaca are the paired inferior wings of unsplit mesoderm whose peripheral margins form the mesodermic borders of the cloacal membrane. The ventral wall or floor of the cloaca is the cloacal membrane. Dorsal to the cloaca are the axial structures of the notochord and neural tube. The cranial wall or roof is formed by the union of the inferior portions of the splanchnopleures, and is the floor of the cœlomic cavity.

The surface cells of the mesoderm lining the interior of the cœlomic cavity become flattened and form the continuous sheet of mesothelium or peritoneum.

PART II

UROGENITAL APPARATUS AND UROGENITAL FOLD—KIDNEYS AND BLADDER—GENITAL GLANDS—UROGENITAL UNION, GROWTH AND DEGENERATION—FINAL CHANGES IN UROGENITAL FOLD WITH CHANGED POSITION OF GENITAL GLANDS

Urogenital Apparatus, Kidneys and Bladder. The dorsal abdominal wall, between the intestinal mesentery and the lateral parietes is the region of the mesoderm of the intermediate cell mass. Faint transverse grooves mark off the longitudinal area into segments. Beginning above, each segment develops an outgrowth of solid mesoderm dorsally to form the pronephros. The outgrowth extends horizontally dorsad, and approaching the ectoderm turns caudad to unite with the caudal portion of the succeeding segment below. The caudal portion of the outgrowth from the terminal segment fuses with the ventrolateral wall of the cloaca near the cloacal membrane. The horizontal outgrowths disappear as their caudal portions unite. The union of the caudal extensions of the mesodermal downgrowths forms a long cord—primary excretory cord—attached to the wall of the cloaca.

The primary excretory cord runs in the mesoderm at first close to the ectoderm and then bends ventrally towards its attachment on the ventromedial portion of the wall of the cloaca close to the cloacal membrane.

The pronephros does not function as an excretory organ. While the pronephros is disappearing, horizontal outgrowths appear from the same parent nephrogenic tissue, and advance directly into the primary cord at its side. The mesonephros, or Wolffian body is formed. A continuous lumen develops in the horizontal outgrowths and their attached long cord and the secretory tubules of the mesonephros communicates by its primary excretory or Wolffian duct with the entodermal cloaca. The simple horizontal tubules become coiled. The wall of the proximal coil becomes invaginated and a vascular tuft grows against this wall to form a Malpighian corpuscle. The mesonephros functions as the provisional kidney of the fetus.

The Wolffian fold of each side lateral to the excretory duct becomes depressed by a cornet-shaped evagination into the fold

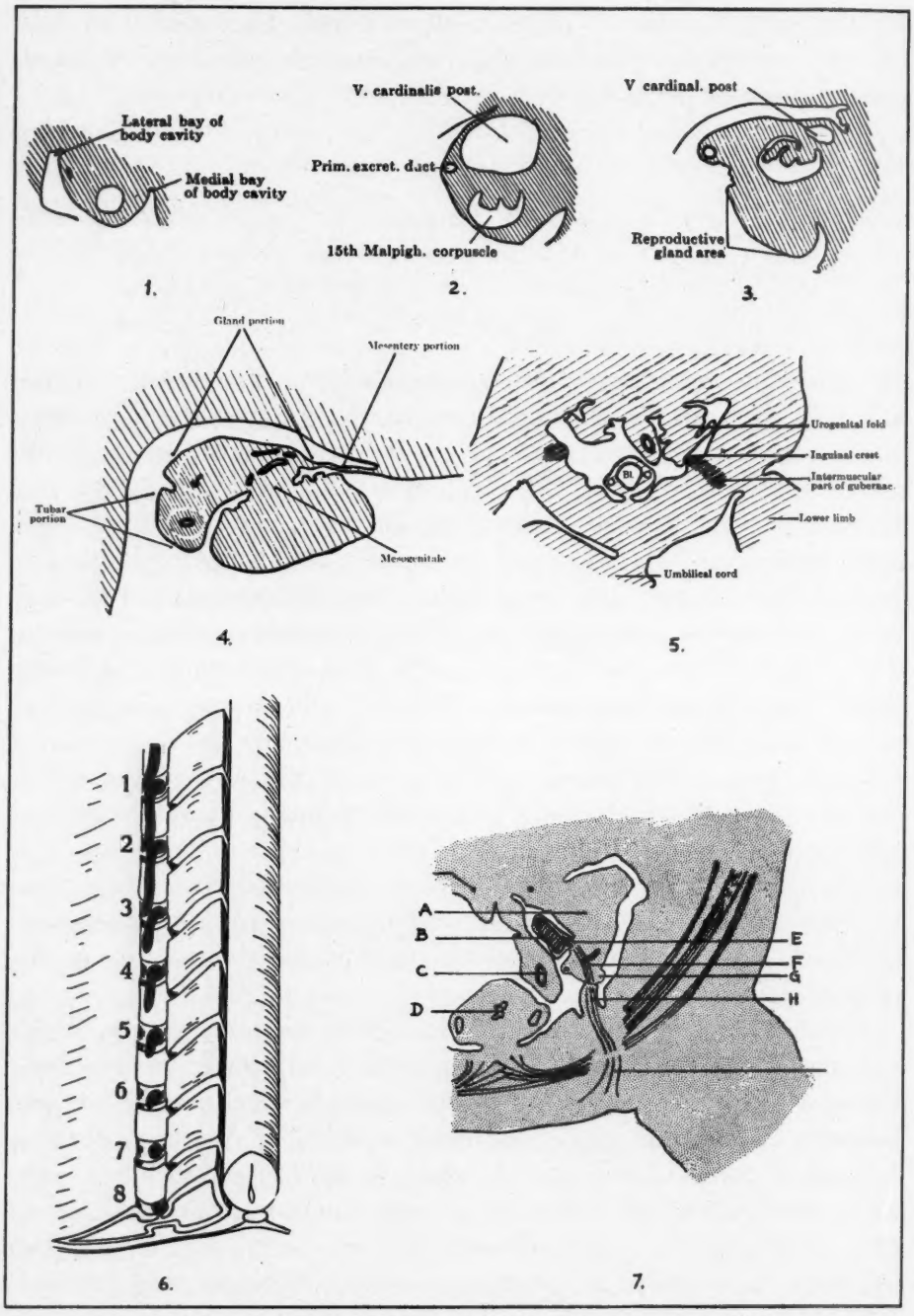


PLATE VIII. 1–5 and 7, transverse sections including body cavity of successive stages of the urogenital fold. 2, embryo of 12.5 mm. (Hochstetter). 6, pronephros—dorsal view showing union of caudal extensions. A, urogenital fold. B, testicle. C, intestine. D, bladder. E, genital ligament. F, inguinal ligament. G, inguinal crest. H, intermuscular part of chorda gubernaculi. Terminal fibers at base of labioscrotal fold as in 5 and 7. (After Felix.)

by a downgrowth of mesothelium from the surface. The downgrowth continues and forms the Müllerian ducts.

The primary excretory duct, before its insertion into the wall of the cloaca, develops a diverticular evagination—ureteric bud—from its dorsolateral wall. The ureteric bud grows dorsocephalad into the surrounding nephrogenic mesoderm. The mesoderm is compressed by the growing bud to form a covering layer for its tip—metanephrogenic cap. The tip of the ureteric bud and its cap of mesoderm form the metanephros or permanent kidney.

The elongating ureter advances the metanephros caudocephalad, deep to the mesonephros. The single ureteral bud undergoes successive divisions which impede the upward course of the kidney. The kidney comes to rest under the enormously growing suprarenal body.

The branching of the ureteral tip divides the metanephric cap into isolated segments. Each segment develops a lumen to form a vesicle. The vesicle elongates into a tubule which becomes convoluted, and, finding by its distal end the extremity of a terminal subdivision of the ureteric tree, unites with it to form a continuous tubular structure that empties into the ventral cloaca. A tuft of vessels follows the invagination of the proximal coil and a Malpighian corpuscle is formed. The permanent kidney is established. The kidney and its ureter are retroperitoneal. Each kidney lies under a portion of the colon and its ureter is crossed by the structures that develop from the urogenital fold.

The lumbodorsal anteflexion of the embryo constricts the middle portion of the urogenital cloaca against the pubis. The cavity of the ventral cloaca expands above as the bladder, narrows at the pubis as the pelvic portion of the cloaca and expands inferiorly as the urogenital sinus. The walls of the ventral cloaca taper above as they pass from the expanded portion into the mesoderm of the umbilical cord that supports the allantoic continuation of the cloacal cavity. The allantois is the principal attachment of the bladder.

The Wolffian duct with its ureteral bud is attached to the bladder. As the bladder expands the ureteric diverticulum is taken up into the expanded wall. The common duct of the ureter and Wolffian duct empties into the cavity. The ureteric orifice in the bladder remains patent. The terminal piece of the excretory duct replaces the related portion of the bladder and the duct opens into the bladder at a lower level and more medially. The bladder, with its laterally placed ureters is carried upward by the cephalad migration of the

umbilical cord. The excretory ducts open into the pelvic portion of the urogenital cloaca on its dorsal surface.

The entodermal wall of the cloaca between the orifices of the ureters and the excretory ducts is replaced by the mesoderm of the excretory ducts. This area of mesoderm is the trigone of the adult bladder.

Urogenital Fold. The development of the mesonephros in the dorsal body wall forms a bulge—urogenital fold—into the cœlomic cavity. The fold is early covered by epithelium spread uniformly in two layers. Later the epithelium on the summit and median slope of the fold to the root of the mesentery becomes many layered, and constitutes the reproductive gland area. This extends throughout the length of the median half of the urogenital fold. Epithelium from the thickened area grows into the fold and compresses its loose mesonephric zone into a strip of denser tissue which becomes sharply defined from its overlying epithelium. The Malpighian corpuscles and mesonephric tubules are displaced dorsally. The growth of the ingrowing epithelium forms a solid epithelial mass—genital gland—with a distinct boundary from the subjacent mesoderm. The epithelial mass separates into a superficial epithelium and epithelial nucleus. When the mass attains a third of the thickness of the urogenital fold the genital gland forms a mound on the fold which becomes marked off in the urogenital fold by a groove at the boundary between the epithelial mass and the Wolffian body that is incomplete above and below. The gonad is free at the sides and continuous with the urogenital fold above, below and by its median portion. In the female, the urogenital union becomes nonfunctioning, but in the male it remains functioning. The portion of the urogenital fold between the fossae of the grooves is the stalk of the genital fold. The outer portion of the mesonephric fold containing the Müllerian tubes is marked off by a groove and the broad attachment of the fold to the dorsal abdominal wall becomes attenuated. The mesonephric fold consists of three portions: the tubar, the common portion of the mesonephric gland and excretory duct, and the dorsal mesentery. The stalk of the genital gland is attached to the mesonephric gland portion of the fold.

General Distinctions of Gonads. The reproductive glands of both sexes develop from the epithelial nucleus of the genital fold. In the male the epithelium forms cords which acquire a connective tissue investment to form the primitive testis. Many of the cords become hollowed out to form the tubules of the testis. The form of the cord and later of the tubule is determined by the indifferent cells from

which they principally originate. The genital cells or spermatogonia have no influence on the form. In the female the epithelial nucleus is passively divided by inwandering connective tissue into a network of masses and cords of cells to form the ovary. Each genital cell forms a unit by itself. The form of the primary follicles is determined by their ova.

The covering layer of the tunica albuginea of the sex glands is formed from the outermost layer of the epithelial nucleus. The portion of the epithelial nucleus that projects into the genital stalk forms the rete cords of the sex gland.

Urogenital Union. The invasion of the urogenital fold by the ingrowing epithelial nucleus of the genital gland is associated with a degeneration of the lower mesonephric secretory tubules as far as their transition into the collecting tubules. The distal ends of the collecting ducts lie in an epithelial bay of the genital rete cords from which they are sharply defined. The cords become tubular and, uniting with the distal ends of the collecting tubules, effect the urogenital union. The collecting ducts are still connected with their excretory—Wolffian—duct.

Male Sex—Changes in the Fold; Development and Degeneration. In the male the collecting ducts that have united with the rete tubules form the efferent ducts of the testis. The testicular piece of duct remains straight while the portion attached to the Wolffian duct becomes coiled. The straight portion is surrounded by firm connective tissue and becomes the comus vasculosi. The coiled portion includes the lower end of the excretory duct to become the canal of the epididymis. The remainder of the Wolffian duct becomes the vas deferens. The vas deferens dilates to form an ampulla from which the seminal vesicle develops. Its narrow horizontal portion, the ejaculatory duct opens into the urethra. The epithelium of the duct is ciliated.

Degeneration of the Fold. The Müllerian ducts in the male begin to degenerate shortly after their union to form the uterovaginal canal on the urogenital cloaca. The portion of the tube between the caudal pole of the testis and the uterovaginal canal breaks down, leaving a very small portion situated between the head of the epididymis and the testis—the hydatid of Morgagni. This remnant usually contains a simple epithelial canal. The uterovaginal canal persists in the broad horizontal portion—vagina masculina. Müller's tubercle persists as the colliculus seminalis. The cristae urethralis are early defined.

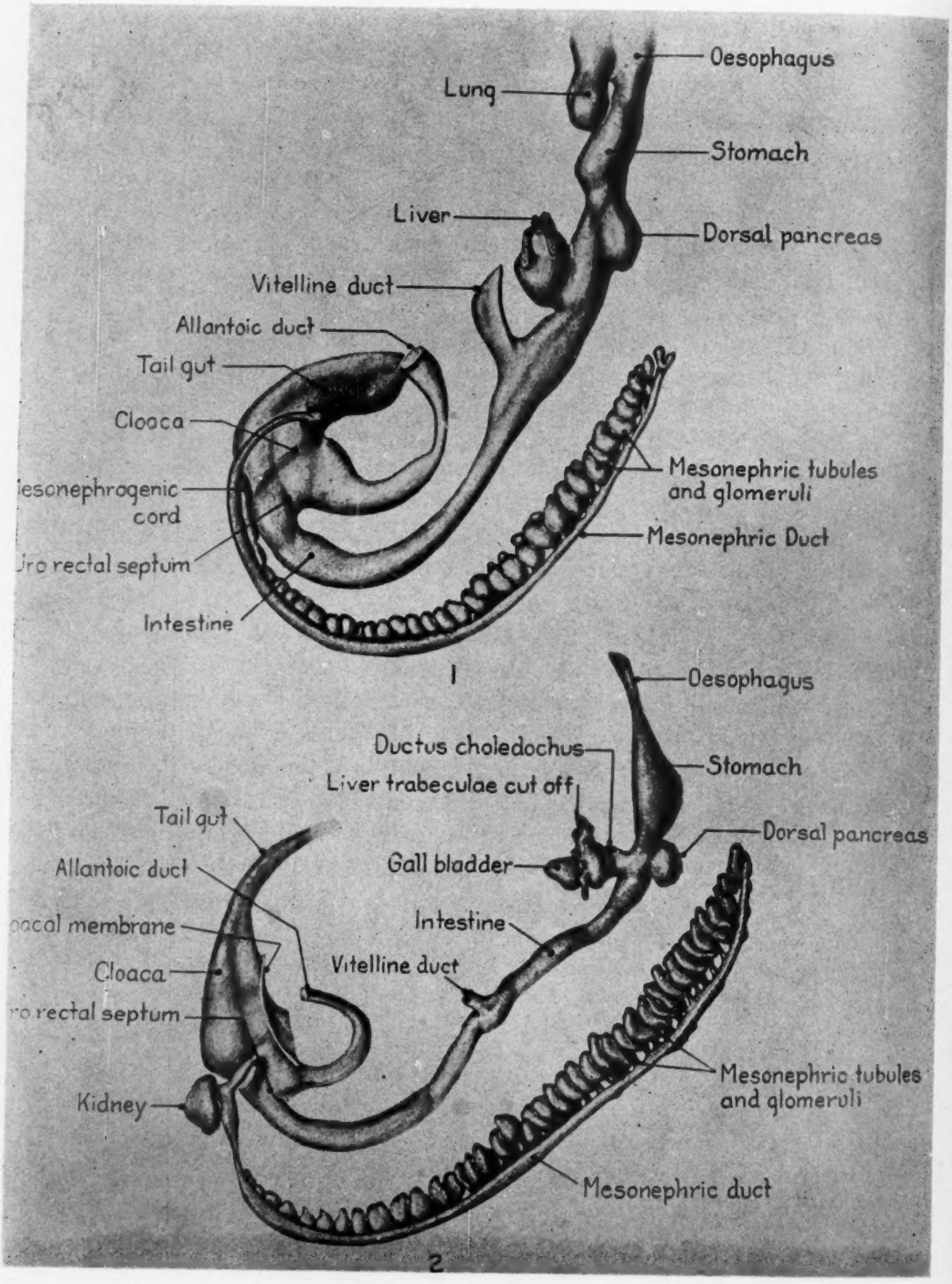
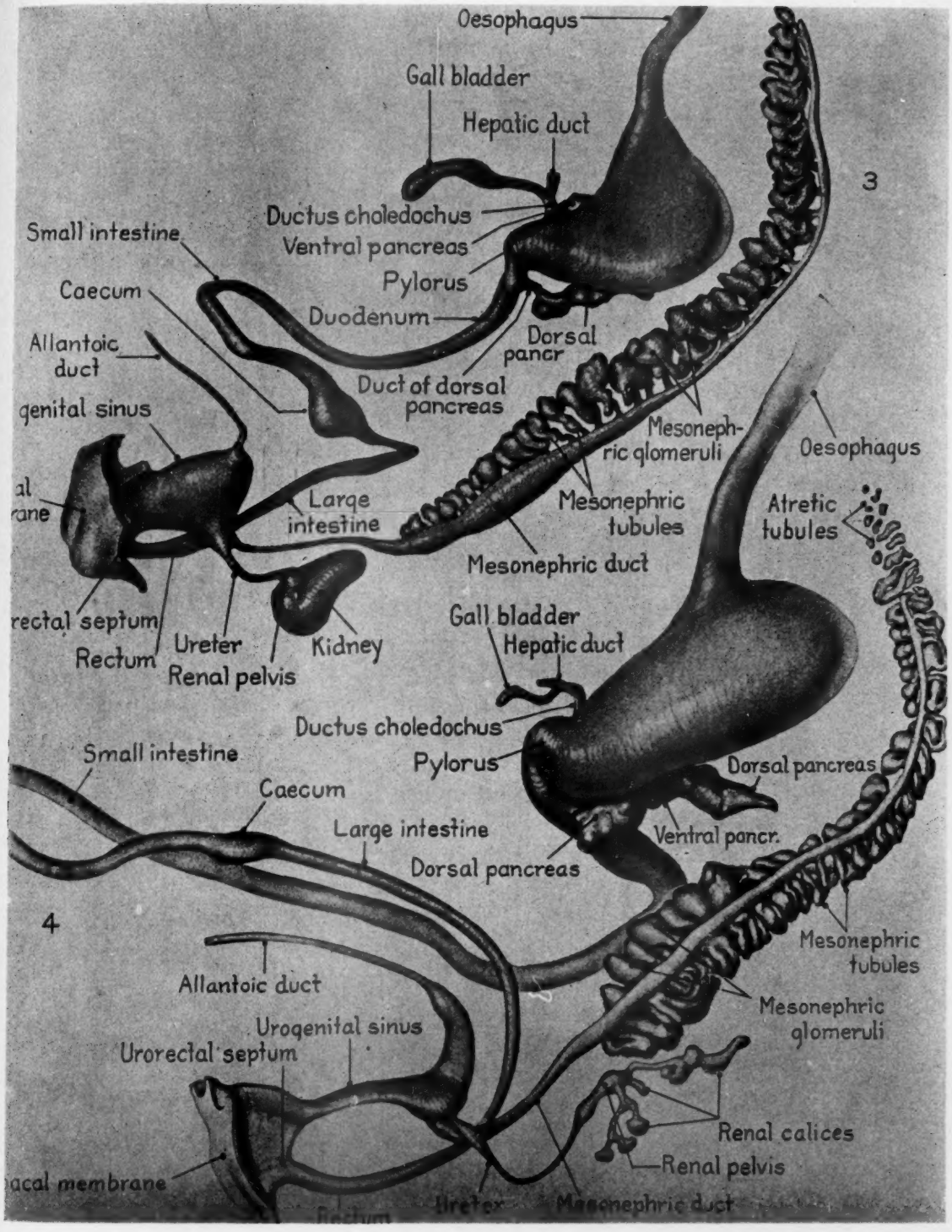


PLATE IX. 1, mesonephros, alimentary canal in 4 mm. human embryo. 2, mesonephros, metanephros embryo. 4, same, showing further development



and alimentary canal of 5.5 mm. human embryo. 3, same as 2, showing further development in 8 mm. in 14.6 mm. embryo. (After Shikinami.)

Mesonephros. Degeneration of the mesonephros begins with the completion of its cranial growth. Proceeding from above down the tubules of the cervical and thoracic segments disappear with their corresponding portion of the Wolffian duct. Later, the remaining tubules, all in the lumbar region are in some stage of degeneration. The upper tubules unite with the sex gland, and the Wolffian remnant consists of an upper genital portion or epigenitalis, and a lower mesonephric gland portion, the paragenitalis. The break in the tubules occurs at the junction of the collecting ducts with the secretory tubules. The mesonephric tubules attached to the Wolffian duct lie in the urogenital fold that has gained attachment to the anterior abdominal wall. The urogenital fold follows the wall and, becoming drawn out, forms a double bend to enter the true pelvis. The Wolffian duct is carried by the fold, and in its horizontal portion receives the paragenitalis. The excretory duct forms a loop that carries its attached collecting ducts away from the secretory tubules and Malpighian corpuscles. The latter structures remain caudal to the epigenitalis and may be present in cranial and caudal groups. The first group of corpuscles and tubules may persist, but usually disappears. In the latter case there is one paragenitalis; in the former there are two.

The collecting tubules carried away with the Wolffian duct may have their individual orifices in the duct reduced to a single orifice which forms the collecting duct of the paradidymis or the canaliculus aberrans Halleri. The paragenitalis forms the paradidymis, or the organ of Giraldis. When present it lies between the head of the epididymis and the testis.

Changes in position of the testis and its duct are influenced by related structures. The testicle as a member of the urogenital fold retains its attachment to the fold and its position is an incident in the changing position and attachments of the fold. As a projection into the body cavity the testis is associated with the peritoneal cavity of its immediate vicinity.

The urogenital fold begins as a bulge from the dorsal body wall that extends longitudinally from the diaphragm to the dorsal wall of the ventral cloaca. The fold at this time is attached by a broad base to the dorsal wall. The lateral wall is narrow, and the anterior wall is non-existent.

As the urogenital structures become defined in the corresponding subdivisions of the fold, the broad base of the fold is defined from its dorsal wall as a stalk which becomes attenuated to form the mesentery of the fold. The mesentery attached by its upper portion

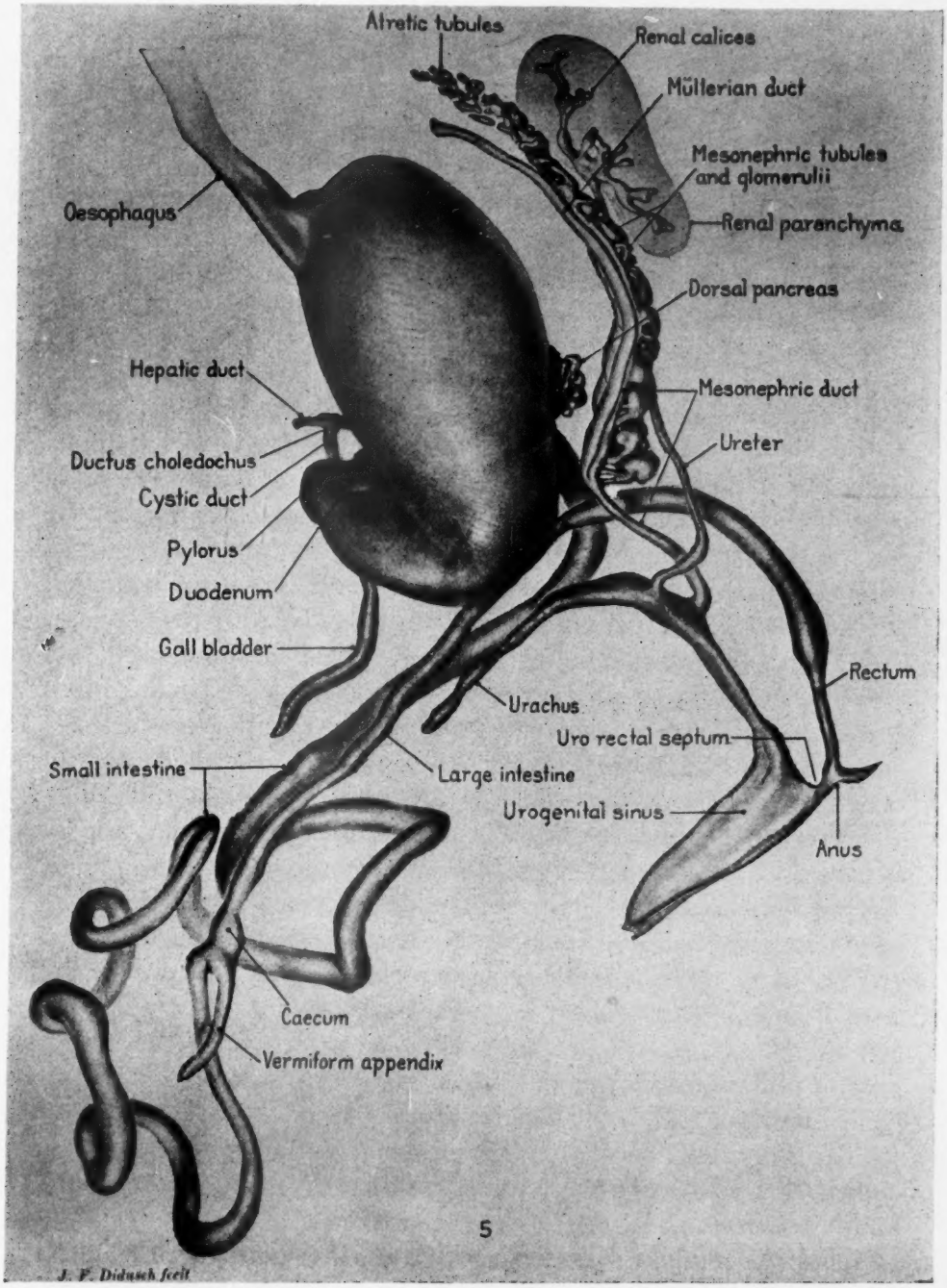


PLATE X. Continuation of Plate IX. 5, same as 4, showing development in 23 mm. embryo. Urorectal septum is the pelvic bar. (After Shikinami.)

to the diaphragm is the diaphragmatic ligament of the mesonephros. The mesenteric ligament keeps pace with the degenerating mesonephros as it becomes reduced from above down and takes progressively lower attachments on the dorsal wall until the spermatic vessels, growing into it, convert the diaphragmatic ligament of the mesonephros into the permanent mesentery of the spermatic vessels. By this time, the mesonephros has collapsed and the testis through its mesorchium has acquired the diaphragmatic ligament of the Wolffian body.

In the beginning the urogenital fold of each side lies parallel to the vertebral column. Organs developing in the midline force the upper portions of the fold away from each other. The enormous growth of the suprarenal bodies and the metanephroi in the dorsal wall spread the folds laterally. The large stomach and intestinal coils further displace the folds towards a horizontal position. Room for the displacement has been provided by the broadening of the dorsal wall. Below the metanephroi, the folds retain their sagittal position. The fold forms a double bend as it lies between its displaced upper and undisturbed lower portions. The bent urogenital fold consists of three portions; an upper sagittal, a middle horizontal, and a lower sagittal. The lower sagittal portions unite with the lower dorsal wall of the ventral cloaca and with each other. The area of fusion is the genital cord and contains the fused Müllerian tubes and the primary excretory ducts. The Müllerian ducts disappear as far as their fused portion which persists as the vagina masculinus. The horizontal portion of the bent fold develops a fold at its outer bend—inguinal fold—which grows towards the dorsum of the lateral wall to unite there with the ridge—inguinal crest—formed in the lateral wall. The horizontal urogenital fold, comprising the medial genital and lateral mesonephros folds, is attached to the lateral wall by the inguinal ligament of the mesonephric fold, and accompanies the wall as it is pushed anteriorly.

The genital fold and its contained gland change their form by cranial degeneration and caudal growth. The gland occupies the fold between its ends. The lower end of the fold—ligament testis—attaches the gland to the lower sagittal portion of the urogenital fold that enters into the genital cord. The caudal pole of the gland, which lies above the true pelvis, becomes constricted off from the mesonephric fold. The ligament of the testis at the junction of the true and false pelvis is attached to the medial portion of the horizontal urogenital fold which is attached to the inguinal crest of the lateral body wall by the inguinal fold.

The inguinal crest, before the mesoderm of the wall has differentiated into muscle, develops a compact cord—chorda gubernaculi—which extends through the thickness of the mesodermal body wall from its apex at the crest to its base near the integument. The musculature of the abdominal wall, as it becomes differentiated from the mesenchyme, grows around the persisting chorda gubernaculi. The cord lies in a canal of the musculature of the abdominal wall. The transversus and the internal oblique provide a mantle of fibers that extend up on the cord to the inguinal crest. The external oblique forms a foramen at which the cord ends by its base. From the foramen a cord of mesoderm forms—ligamentum scroti—that extends to the deep surface of the labioscrotal folds on the perineum. The aponeurosis of the external oblique grows around the ligamentum scroti and forms the cremasteric fascia. The cords of the ligamentum testis, inguinal folds, chorda gubernaculi, and ligamentum scroti unite to form a continuous cord that extends from the caudal pole of the testis, through the inguinal canal to the scrotal sac.

The caudal pole of the testis, the attached mesonephric fold and the gubernaculum at first project into the center of the portion of the abdominal cavity cut off from the main space—saccus vaginalis—and are covered with peritoneum both medially and laterally. Later, the saccus covers only the medial protruding portion when the lateral surfaces of the urogenital fold fuse with the lateral wall of the saccus. Thickening of the anterior abdominal wall fails at the point where the gubernaculum traverses the wall and the developing musculature grows around a small portion of the saccus. The saccus penetrates the musculature and enters the scrotal sac. A narrow peritoneal canal connects the saccus with the abdominal cavity. Shortly after birth the canal becomes solid. The saccus, completely cut off from the abdominal wall, becomes the tunica vaginalis propria of the testis which has wandered through the canal into the scrotal sac.

Female—Degeneration of the Urogenital Union. Changes in the structures of the urogenital folds of the differentiated ovary correspond in general to the changes that take place in the male, with the important differences that the excretory duct disappears in the female and the growing uterus intercepts the caudal ligament of the genital gland.

The mesonephros degenerates in its cranial portion and consists of the upper epigenitalis—epoöphoron—attached to the rete ovarii and the lower paragenitalis—paroöphoron—of the remaining

mesonephric gland. The paroöphoron quickly degenerates in extra-uterine life.

The primary excretory duct, the functionless duct of the ovary, degenerates before the rete ovarii has united with the mesonephros. The epoöphoron is free when it develops. The degeneration of the Wolffian duct takes place at the same time that the Müllerian duct disappears in the male. The excretory duct generates in its portion between the caudal pole of the ovary and the beginning uterovaginal canal. The remains of the excretory duct constitute Gartner's canal. The canal may retain connection with the epoöphoron for a time. The canal first lies in the broad ligament, then sinks into the musculature of the uterus at the internal os, runs through the musculature to pass through the portio vaginalis into the lateral fornix of the vagina. The lower portion passes through the wall of the vagina and, passing through the vestibular epithelium, opens to the exterior near the free border of the hymen. Portions of the canal are present in one fourth of females. The persisting Gartner's canal may form an ampulla and a seminal vesicle in the upper part of the portio vaginalis.

Changes in position of the ovaries and their ducts are influenced by related structures. The ovary, as a member of the urogenital fold, retains its attachments to the fold and its final position is an incident in the changing position and attachments of the fold.

The urogenital fold begins as a bulge of the dorsal body wall into the peritoneal cavity and extends longitudinally from the diaphragm to the dorsal wall of the ventral cloaca. The fold at this time is attached by a broad base to the dorsal wall. The lateral wall is narrow and is yet to form the anterior wall. The structures in the urogenital fold occupy corresponding divisions of the subdivided fold which are connected to each other. The broad base of the urogenital fold at its cranial end becomes a thready mesentery, the diaphragmatic ligament of the mesonephric fold. The ovary occupies the genital fold, and the remainder of the urogenital fold is divided into the tubar portion for the Müllerian tubes, the gland portion for the mesonephros and its excretory duct, and the diaphragmatic ligament of the mesentery. The mesonephric fold joining the tube and ovary to the diaphragmatic ligament degenerates and the developed ovary and its tube acquire the mesenteric portion of the urogenital fold. The mesentery forms divisions for the tube, infundibulopelvic ligament, and for the ovary, the ovaricopelvic ligament. The ligaments, thickened with the ingrowth of blood vessels,

retain their connection with the degenerated mesonephric gland portion of the urogenital fold.

The urogenital folds, at first parallel to the vertebral columns, are displaced laterally above the true pelvis by the medial suprarenals and metanephroi of the dorsal body wall and the stomach and intestines of the abdominal cavity. Each fold forms a double bend between the upper sagittal and lower sagittal portions. Between the two bends is the horizontal portion.

The lower sagittal urogenital folds unite first with each other in the floor of the pelvis to form the ventral partition of the cavity, and then with the inferior dorsal wall of the ventral cloaca. The union of the folds forms the genital cord. The genital cord divides the pelvic cavity into the recto-uterine and vesico-uterine fossae. The upper end of the genital cord lies at the level of the second bend of the fold.

Within the genital cord, the Müllerian ducts lie between the excretory ducts and the whole mass forms a projection of the dorsal wall of the vesico-urethral sinus to form the Müllerian tubercle on its interior. The Müllerian ducts grow to the tubercle and then run horizontally on the wall of the sinus. The horizontal portion of the ducts is solid. The upward growth of the vesicle portion of the ventral cloaca leaves the Müllerian tubercle as a projection on the dorsal wall below it.

The right and left Müllerian tubes unite by their median external surfaces within the genital cord and form the paired uterovaginal canal. The attached median walls fuse to form a septum which becomes absorbed in the caudocranial direction to form the single uterovaginal canal. The Müllerian tubes, at their abdominal origin of a single funnel, acquire secondary funnels. The tubes on their completion of the uterovaginal canal run vertically downwards in the lateral tubar portion of the mesonephric fold, to the edge of the genital cord, bend there across the excretory ducts to run horizontally towards the midline to unite in the uterovaginal canal. Each Müllerian tube consists of a vertical, a horizontal and a fused portion in the uterovaginal canal. The vertical portion becomes the tube of the adult; the horizontal portion forms its half of the fundus of the uterus; and the fused portion the cervix and upper four fifths of the vagina. Interference with the union of the two tubes by the ventral structures of the body wall results in various anomalies of the uterovaginal canal.

The horizontal piece of the tube lies in the tubal portion of the urogenital fold at the level where the fold develops its inguinal

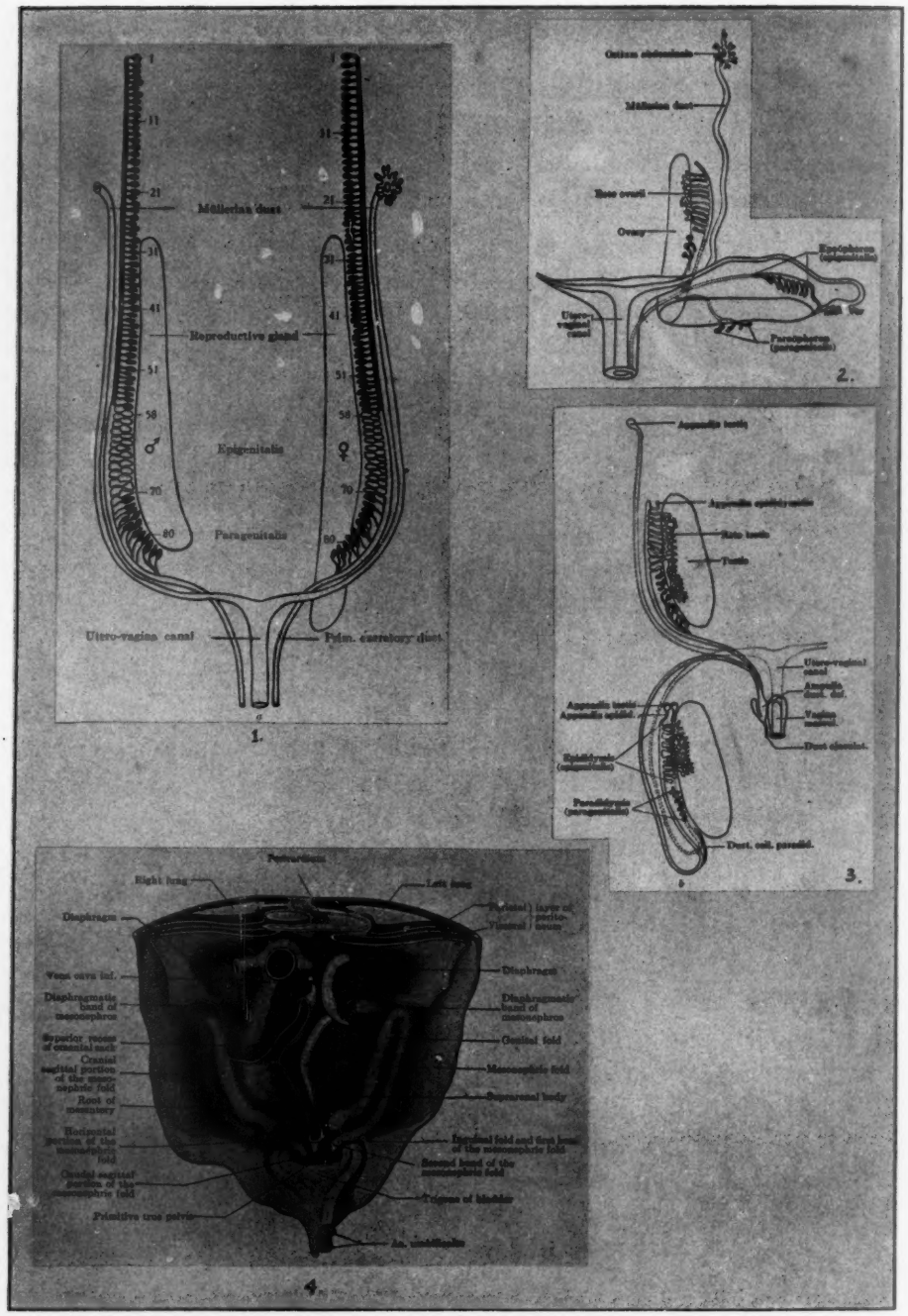


PLATE XI. Urogenital fold and ultimate disposition of its structures. 1, diagrammatic representation of the entire mesonephric organ, reproductive gland, and efferent ducts. Left is male and right is female reproductive gland. 2, after migration of the ovary. Degenerated structures in dotted lines. 3, after migration of the testis. Degenerated structures in dotted lines. 4, model of posterior wall of human embryo of 19.4 mm. (After Felix.)

ligament that attaches to the gubernaculum of the abdominal wall.

The genital fold and its ovary change their form and position by cranial degeneration and caudal growth. The caudal pole of the ovary reaches the true pelvis behind the genital cord of the united mesonephric folds. The ovary occupies the fold between its ends. The lower end of the genital fold—ligament of the ovary—attaches the caudal pole of the ovary to the genital cord. The pathway of the ovarian ligament through the horizontally bent urogenital fold to the inguinal ligament for the abdominal wall is interrupted by the growth of the uterus from the horizontal portion of the Müllerian tube. The ovarian and inguinal ligaments growing to each other are intercepted by the uterus to which each gains an attachment. The inguinal ligament from its subserous attachment to the uterus unites with the chorda gubernaculi of the abdominal wall—round ligament of the uterus—which continues as the ligamentum labiale to the deep surface of the integument of the labial fold of the external surface. The inguinal canal and the peritoneal extension into the canal, form as in the male.

The ovary rotates around the caudal pole through an angle of 90 degrees and its cranial pole takes a lateral position. The tube portion of the Müllerian duct is carried laterally by the rotation of its attachment to the dorsal abdominal wall, the attenuated base of the original urogenital fold, and takes a lateral position above the ovary. The cranial portion of the tube is in relation to the cranial portion of the ovary during development and after rotation.

The urogenital sinus, at each side of its posterior wall, develops evaginations—sinovaginal bulbs—which extend to the Müllerian tubercle and fuse with it. The bulbs become solid by the proliferation of their lining epithelium and fuse with the tip of the Müllerian—uterovaginal—ducts which become solid by the same process. The solid mass of cells is the vaginal plate which grows in all directions. The central cells of the solid vagina break down to form the cavity of the vagina. The cranial end of the vagina is defined by the formation of the anterior and posterior fornices as solid epithelial projections. The caudal end expands and pushes in the posterior wall of the urogenital sinus and extends along the posterior wall to form the caudal segment of the hymen. The anterior paired segment of the hymen is formed where the evaginations of the urogenital sinus occur to form the sinovaginal bulbs. The vagina is formed in its lower one fifth from the sinovaginal bulbs which arise from the epithelium of the urogenital sinus.

PART III

ENTODERMAL CLOACA—DIVISION PELVIS, PELVIC BAR, UTEROVAGINAL CANAL, BLADDER, URETHRA, GLANDS OF UROGENITAL SINUS

Entodermal Cloaca. The cloacal membrane, which provides the sites of the orifices on the surface of the caudal end of the body, the future perineum, appears before the primitive streak is completed. At this time the membrane is the small area of the caudal surface of the embryonic disc where the ectoderm and entoderm are most intimately apposed.

The ectodermal amniotic sac grows posteriorly against the body stalk and eats its way through the morula mesoderm of the stalk to the entodermal lining of the allantois. The area of ectoderm closely applied to entoderm has been extended from the cloacal membrane of the embryonic plate for a short distance on the dorsum of the body stalk. The mesoderm of the embryonic plate condenses into sheets with superior and inferior wings. The inferior wings of mesoderm run along the lateral borders of the extended cloacal membrane and lose themselves in the morula mesoderm of the body stalk. The axial structures grow down to the upper angle of the membrane. The tail fold forms, and the cloacal membrane extends ventrally from the posterior termination of the cloaca to the body stalk. At this stage the membrane lies in the first plane of the perineum. The lateral borders of the membrane are the medial margins of the inferior wings of mesoderm. The cavity enclosed by the tail fold is the entodermal cloaca which empties in the allantois of the stalk. The inferior wings compose the lateral walls of the cloaca and, when the mesoderm splits to form the body cavity and the intestinal tube, remain unsplit. The cloaca becomes closed off from the abdominal cavity by the union of the lowermost splanchnopleure. The cloaca is the expansion of the hind gut, and terminates superoventrally in the allantois of the stalk. The partition of the fused inferior splanchnopleures separates the cavity of the cloaca from the abdominal cavity. The ventral wall of the cloaca is the cloacal membrane. The cloaca occupies the future pelvis. The partition between the cloaca and the abdominal cavity sags along the ventrolateral borders of the hindgut. The abdominal cavity is extended inferiorly to form the pelvic cavity by the sagging partition.

The caudal portion of the embryo grows beyond its junction with the cloacal membrane to form the tail. The tail carries out a portion

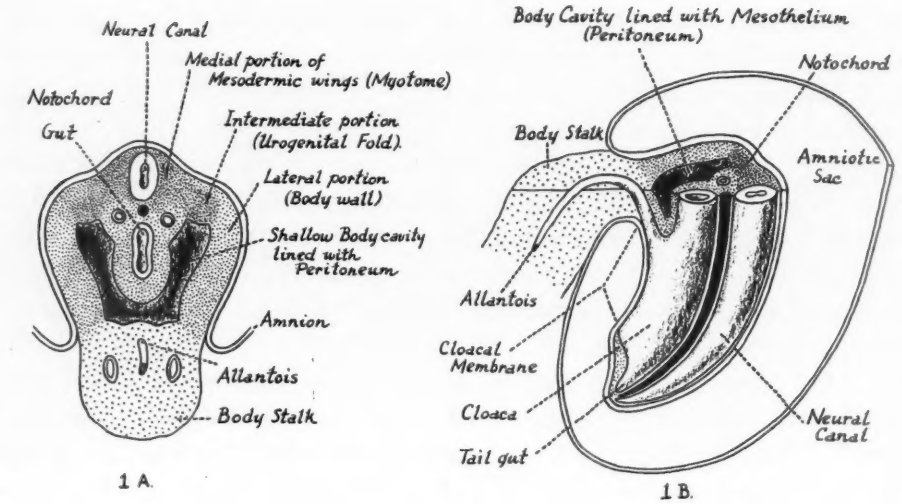
of the cloacal cavity as the tail gut, which soon disappears. The tail curves ventrally above the cloacal membrane toward the body stalk and the perineum is carried forward to face cranially. The tail curves backwards on itself and gradually degenerates. The recurving tail draws the perineal plane forward and downward. At this time the lowest point of the sagging partition has arrived by its entodermal layer at the entodermal layer of the cloacal membrane, and the two layers fuse. The partition is splanchnopleure composed of mesoderm lined on its abdominal surface by peritoneum, and on its cloacal surface by entoderm.

The migration downwards of the splanchnopleure forms the extension inferiorly—pelvic cavity—of the peritoneal abdominal cavity. The attachment of the partition to the deep surface of the cloacal membrane divides the cloacal cavity into two separate portions, the large ventral urogenital cloaca, and the smaller dorsal rectal cloaca. The rectal cloaca is continuous above with the hindgut. The urogenital cloaca receives the Wolffian duct with its ureteric diverticulum, and terminates superoventrally in its allantoic diverticulum in the body stalk. The posterior wall of the ventral cloaca and the anterior wall of the dorsal cloaca are formed from the splanchnopleure and are lined with peritoneum. These walls are joined to each other by the portion of splanchnopleure that has become attached to the cloacal membrane and forms the urorectal septum or pelvic bar. The pelvic bar is the inferior wall of the pelvic cavity, and separates the cavity from the cloacal membrane of the body surface.

The lateral walls of the cloacal divisions are the inferior wings of mesoderm that have remained unsplit, and their medial margins are the mesodermal borders of the cloacal membrane. The cloacal membrane is the ventral wall of the ventral and dorsal cloacae.

The ventral cloaca is constricted at its middle portion by the forward bending of the caudal embryo. The urogenital cloaca forms

PLATES XII, XIII and XIV show the development of the cloaca. Pelvic structures developed from internal (entodermal) cloaca in relation to perineal structures developed from the external (ectodermal) cloaca. The spaces formed as a result of the development of the pelvic structures are separated from the exterior by various portions of the perineum. (1) The prevesical space, between the bladder and the abdominal wall and pubic bone, is separated from the exterior by the tissue enclosing the dorsal vein of the clitoris or the penis. (2) The prerectal space is separated by the contiguous pelvic and perineal bars. (3) The retrorectal space is separated by the contiguous attachments of the rectococcygeus, levator and external sphincter ani muscles. (4) In the female, the vesicovaginal space is separated by the common distal wall of the urethra and the vagina. The prerectal space is similar to the male. These relations are significant in the surgical anatomy of the pelvis and perineum.



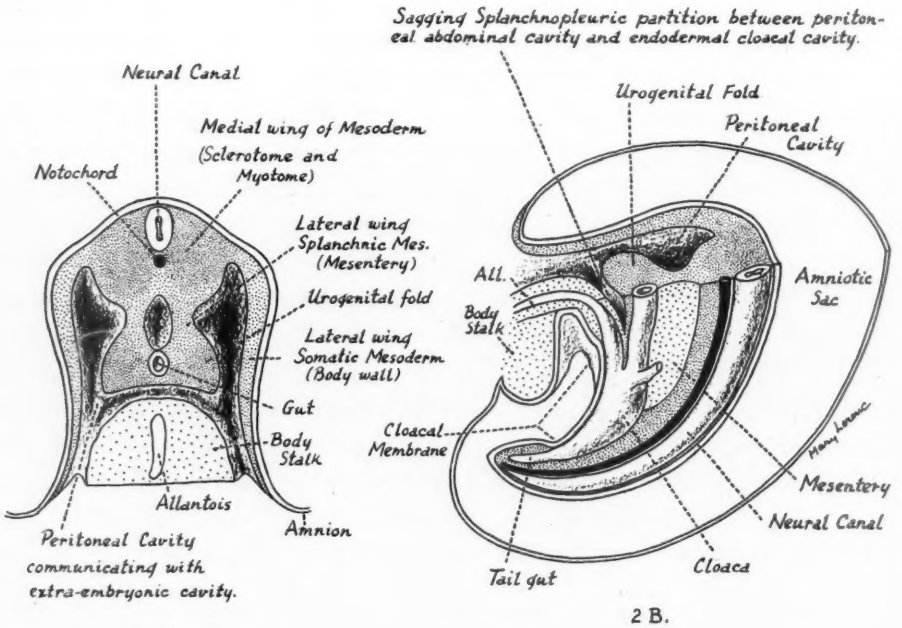


PLATE XII. Three dimensional sections of embryo in region of cloaca. 1A and 2A, cross sections of entodermal cloaca and gut-surrounding mesoderm. Shows peritoneal surface of entodermal cloaca. Shallow peritoneal cavity. 1B and 2B, sagittal sections, entodermal cloaca being divided into urogenital cloaca and rectal cloaca by sagging splanchnopleuric partition.

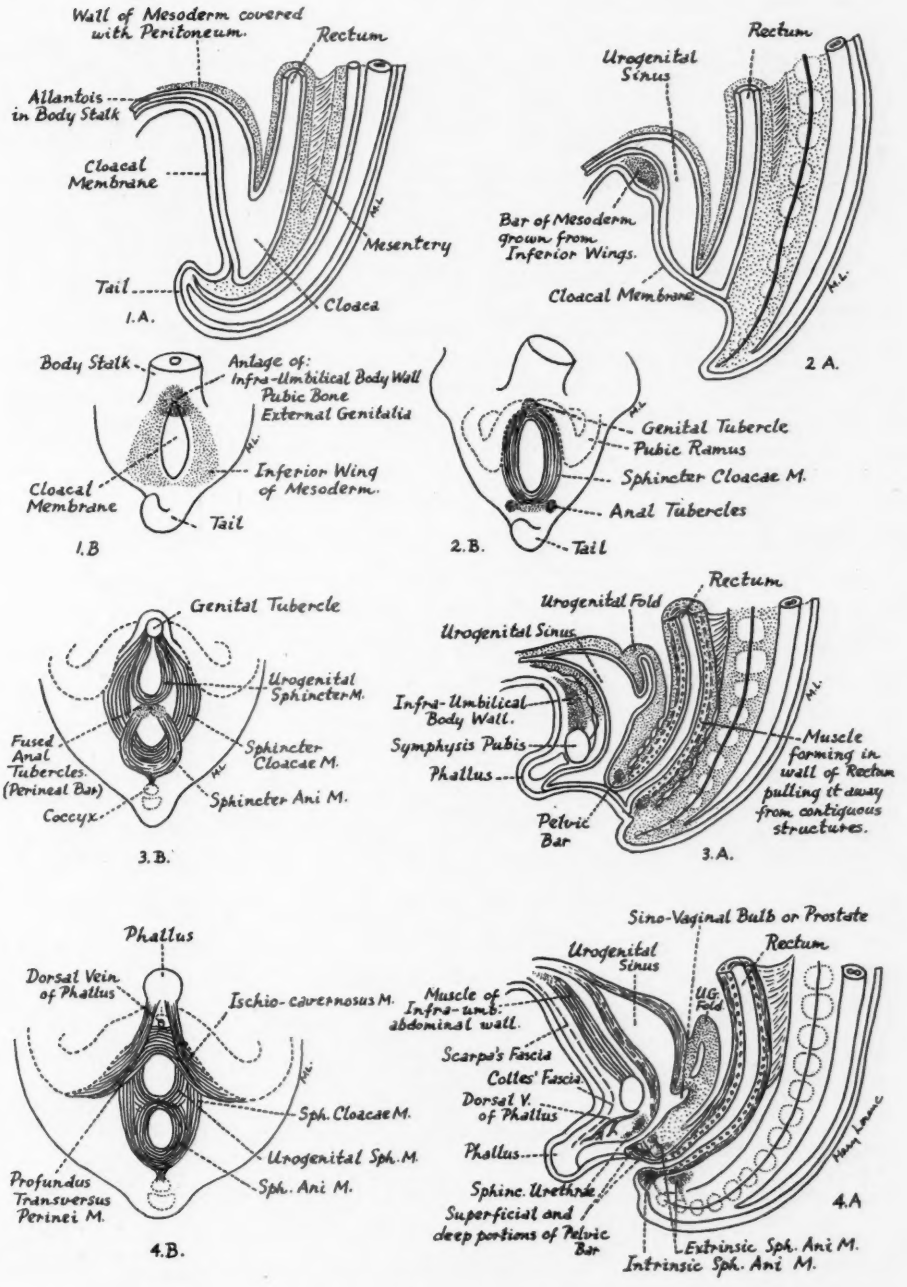


PLATE XIII. Synchronization of entodermal cloacal development and differentiation of mesodermal borders of cloacal membrane. A1–4, sagittal sections of caudal portion of body. B1–4, front views of cloacal membrane showing inferior wings of mesoderm and structures developing from them. 1B, distal extremities of inferior wings of mesoderm forming band at upper extremity of cloacal membrane where it extended on the body stalk. 4A, *UG fold* is the genital portion of the urogenital fold where the uterovaginal canal has formed.

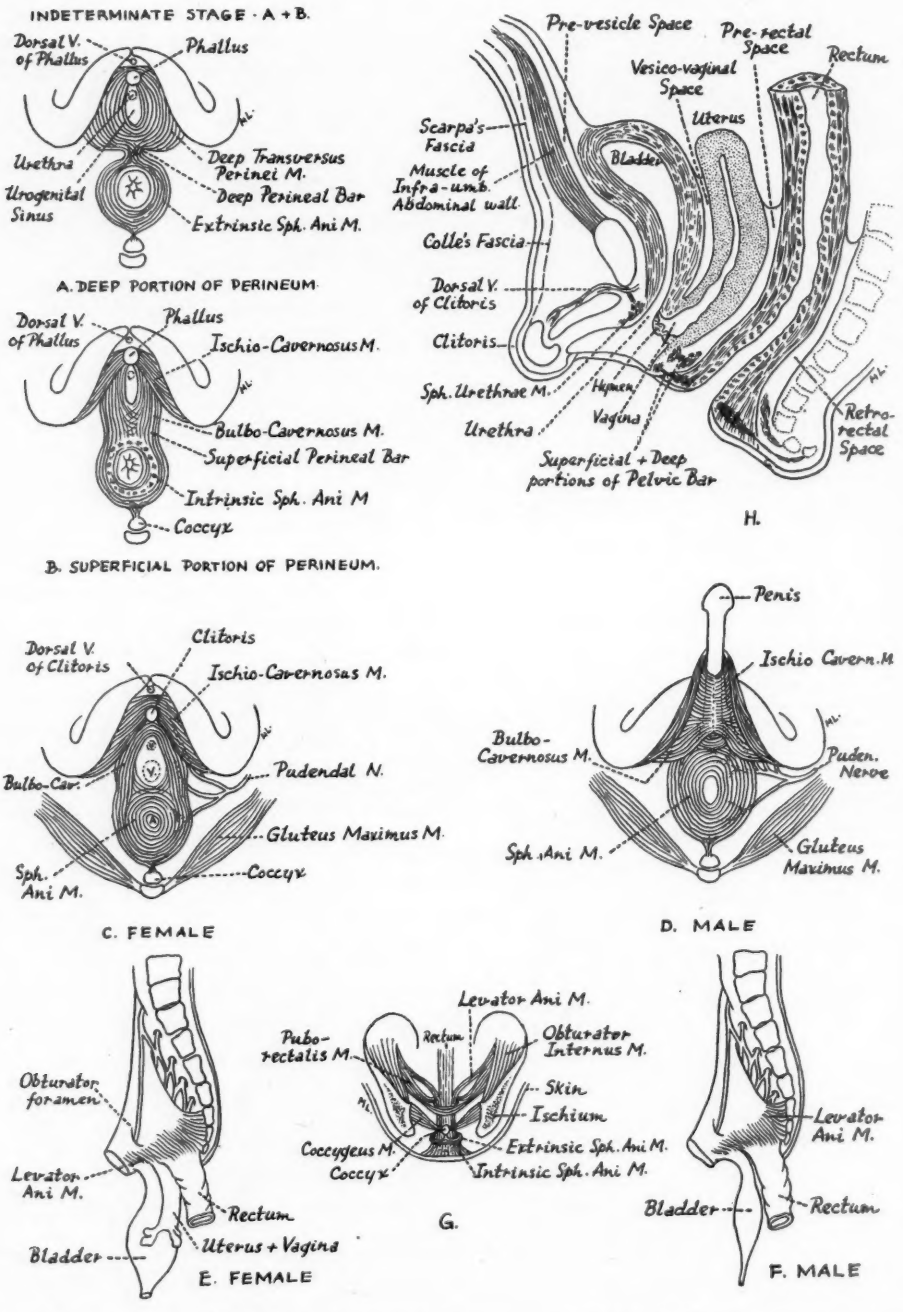


PLATE XIV. Completed pelvic and perineal structures. A and B, indeterminate stage—perineal musculature, deep and superficial portions, front view. C and D, perineal musculature, female and male, front views. E and F, sagittal sections, female and male pelves. G, longitudinal muscle of rectum in relation to levator muscle, external sphincter ani and skin, showing extrinsic and intrinsic levator and sphincter ani. H, relations of the peritoneal lining of the pelvic cavity. Retroperitoneal cavities of the pelvis. Structures separating cavities from the exterior.

an expanded upper portion, the bladder, connected to the allantois; a narrow middle portion, the pelvic urethra; and an expanded inferior portion, the urogenital sinus. The sinus is separated from the exterior by the cloacal membrane. The bladder wall takes in the ureteral orifices and carries them away from the orifices of the excretory ducts as its attached allantois is carried upwards by the cephalad advances of the umbilical cord. The excretory ducts open into the pelvic urethra.

The uterovaginal canal built in the dorsal wall of the pelvic urethra presses its peritoneal wall against the anterior peritoneal wall of the inferior portion of the rectum. The peritoneal walls become applied to each other, and the recess is reduced to a potential cavity. The peritoneum of the reduced pelvic fossa spreads over its gap leading into the potential space and closes it over. The space extends from the pelvic peritoneum above to the pelvic bar below and lies between the rectum and vagina in the female; in the male between the rectum and prostate. The growth of the prostate posteriorly or the growth of the vagina posteriorly, compresses the anterior wall to the posterior walls of the space, and they fuse to form a thickened sheet—Denonvillier's fascia or prostatoperitoneal aponeurosis or vaginal peritoneal aponeurosis, respectively. The active muscular wall of the rectum frees itself from the peritoneal aponeurosis which remains applied to the vagina or prostate. The rectum forms a space —prerectal space—with the peritoneal aponeurosis in front, anterior rectal wall behind, pelvic peritoneal floor above, pelvic bar below. The pelvic bar separates the prerectal space from the cloacal membrane of the external surface. The peritoneal lining separates the pelvic cavity from the prerectal space.

In the female pelvis the uterovaginal canal forms the uterovesical fossa and the uterorectal fossa. The upper four fifths of the vagina is in relation with the dorsal wall of the bladder and proximal urethra.

The urogenital sinus forms the lower fifth of the vagina by evaginations of its posterior walls. The anterior wall of this portion of the vagina is continuous with the dorsal wall of the urethra. Above the level of this attachment the anterior wall of the vagina is apposed to the bladder. Their walls do not fuse. The relation of the vagina to the bladder is similar to the arrangement with the rectum.

GLANDS IN THE UROGENITAL SINUS

The bladder, together with the accompanying umbilical arteries, lies completely within the anterior abdominal body wall. The blad-

der, by expanding and elongating, and by the increased size of the umbilical arteries, is crowded out of the abdominal wall; it develops a ventral mesentery and projects into the body cavity. The two umbilical arteries bound a triangular area on the bladder, vesical plate, whose apex is formed by the umbilicus. Within this area the bladder reaches the umbilicus by its cranial apex with which it connects the remains of the allantois; the internal orifice of the urethra lies at the level of the symphysis. The apex of the bladder diminishes in diameter and finally becomes a narrow tubular extension—urachus—of the bladder. Usually the urachus loses its lumen and degenerates, and is reduced to an epithelial cord. The epithelial cord becomes divided into separate portions which may vanish completely before birth. Persistent remains of the urachus may enlarge in a vesicular manner and form urachal cysts. While the epithelial cord disappears, the surrounding connective tissue persists as a cord and forms the attachment of the bladder—medial vesico-umbilical ligament—of the adult.

The bladder, while it lies in the triangular area between the two umbilical arteries, retains its mesentery; it loses its mesentery when it gradually sinks down after birth and comes to lie in the true pelvis.

Prostate. Above and below the opening of the primary excretory ducts, several independent glands grow out into the surrounding denser mesoderm of the wall. In the male the mesoderm develops smooth muscle fibers and dense connective tissue which mark off the structure—prostate—from the surrounding tissue, and enclose the developing glands. In the female the development of the mesoderm into smooth muscle fibers and compact connective tissue does not take place, and the prostatic tubules remain isolated structures. Few glands are formed in the female; later they may undergo development and form the paraurethral glands.

Prostatic glands begin as solid buds around the periphery of the sinus, most numerous on the dorsal surface, less so on the lateral, and rare ventrally. Simultaneous with their appearance those on the dorsal and lateral surface branch, while those on ventral surface remain unbranched and for the most part degenerate. A bilateral symmetry is apparent. Direction is cranial, only lowermost of lateral grow caudally. Two thirds of glands arise caudal to the openings of the primary excretory ducts. The prostatic glands grow into the muscle and force the muscle fibers apart, and so produce the trabecular arrangement of the musculature.

All the prostatic glands are not united in the prostate; accessory glands appear in the interval between the prostate and Cowper's glands.

Bulbo—Urethral and the Vestibular Glands (Cowper, Bartholin). Solid epithelial buds arise from the dorsal wall of the pars pelvina of the urogenital sinus. They are usually paired, two on each side; frequently one on one side and two on the other. The buds of each side grow upward from the sinus and lie at first in the compact mesoderm which forms the corpus cavernosum urethrae. The glands grow through this mantle and enlarge in the looser mesoderm between the rectum and sinus.

In the female, the glands originate as in the male with increased development later. Up to and after birth there is a slow increase of the terminal vesicles, and after puberty another rapid growth. At the climacterium Bartholin glands undergo degeneration and may be wanting in old age.

Small Sinus Glands. Glands are entodermal in origin; they appear in succession around the entire periphery of the urogenital sinus. They possess the same character as Cowper's glands except that they do not attain to the same extensive development.

PART IV

CLOACAL MEMBRANE AND ITS MESODERMAL BORDERS—BAND ON BODY STALK, EXTERNAL GENITALIA, INFRA-UMBILICAL ABDOMINAL WALL—PERINEAL MUSCULATURE, EXTERNAL CLOACA, ANAL ORIFICE, UROGENITAL ORIFICE

Cloacal Membrane and Its Mesodermal Borders. The inferior wings of mesoderm by their medial borders form the mesoderm at the lateral margins of the cloacal membrane. The remainder of the wings form the lateral walls of the internal cloaca. The mesoderm in the region of the cloacal membrane on the under surface of the umbilical cord invade this portion of the membrane from each side and unite to form a band on the under surface of the cord. The mesodermal borders of the reduced cloacal membrane become thickened and raised above the surface to form a trough with the cloacal membrane as its roof.

The band on the cord by its cranial slope forms the beginning of the infra-umbilical abdominal wall. The cephalad advance of the cord and the medial advance of the lateral abdominal wall complete the wall. The caudal slope of the band develops a projection—the genital tubercle—which carries out with it a portion of the urogenital sinus—phallic sinus. The genital tubercle is part of the band on the cord and develops in the wall derived from the band. This accounts for the superficial perineal pouch of the adult.

The genital tubercle at each side slopes into the borders of the trough which lies for a short distance on the under surface of the tubercle. The trough is the genital fossa and its borders are the genital folds. The mesoderm surrounding the reduced cloacal membrane and tubercle forms the sphincter cloacae.

The cloacal borders behind the cloacal membrane develop a tubercle—anal tubercle—at each side, which are joined by a transverse bar across the midline.

The band on the cord at the beginning of its cranial slope develops the pubic bone which forms the anterior pelvic brim and divides the perineum from the anterior abdominal wall. The band develops the musculature of the anterior wall of the bladder. This wall develops extraperitoneally and remains so throughout life.

The anal tubercle at each side advances along the lateral mesodermal border of the cloacal membrane and bends medially to meet its fellow of the opposite side. The anal tubercles fuse on the cloacal membrane at a point corresponding to the dorsal surface of the pelvic bar. The fusion of the tubercles takes place a short distance in front of the cloacal membrane. The membrane is divided by the fused tubercles into a ventral urogenital portion and a dorsal anal portion. The genital fossa is similarly divided into the urogenital portion in front and the anal pit behind. The fused tubercles form the perineal or external bar which lies on the back of the pelvic bar. The prerectal space is separated from the external surface of the perineum by the perineal bar and its contiguous pelvic bar. The sphincter cloacae, by the fusion of the tubercles, forms the urogenital sphincter and the external anal sphincter around the corresponding parts of the genital fossa. The urogenital sphincter develops in conjunction with the genital tubercle, and the anal sphincter in connection with the longitudinal muscle of the rectum.

The longitudinal muscle of the rectum grows beyond the circular muscular coat and penetrates the anal sphincter to gain attachment on the deep surface of the dermal layer. The sphincter cloacae by its anal portion is partly brought into the gut wall and by its urogenital portion into the inferior portion of the infra-umbilical wall. The two portions of the sphincter cloacae remain attached to each other throughout life. The sphincter cloacae is composed of deep and superficial portions.

The levator musculature develops with the pelvic bones, the tail, and the rectum. Secondary attachments are formed with contiguous structures; urethra, prostate, vagina. A portion of the levator musculature attached to the rectum separates on its perineal surface

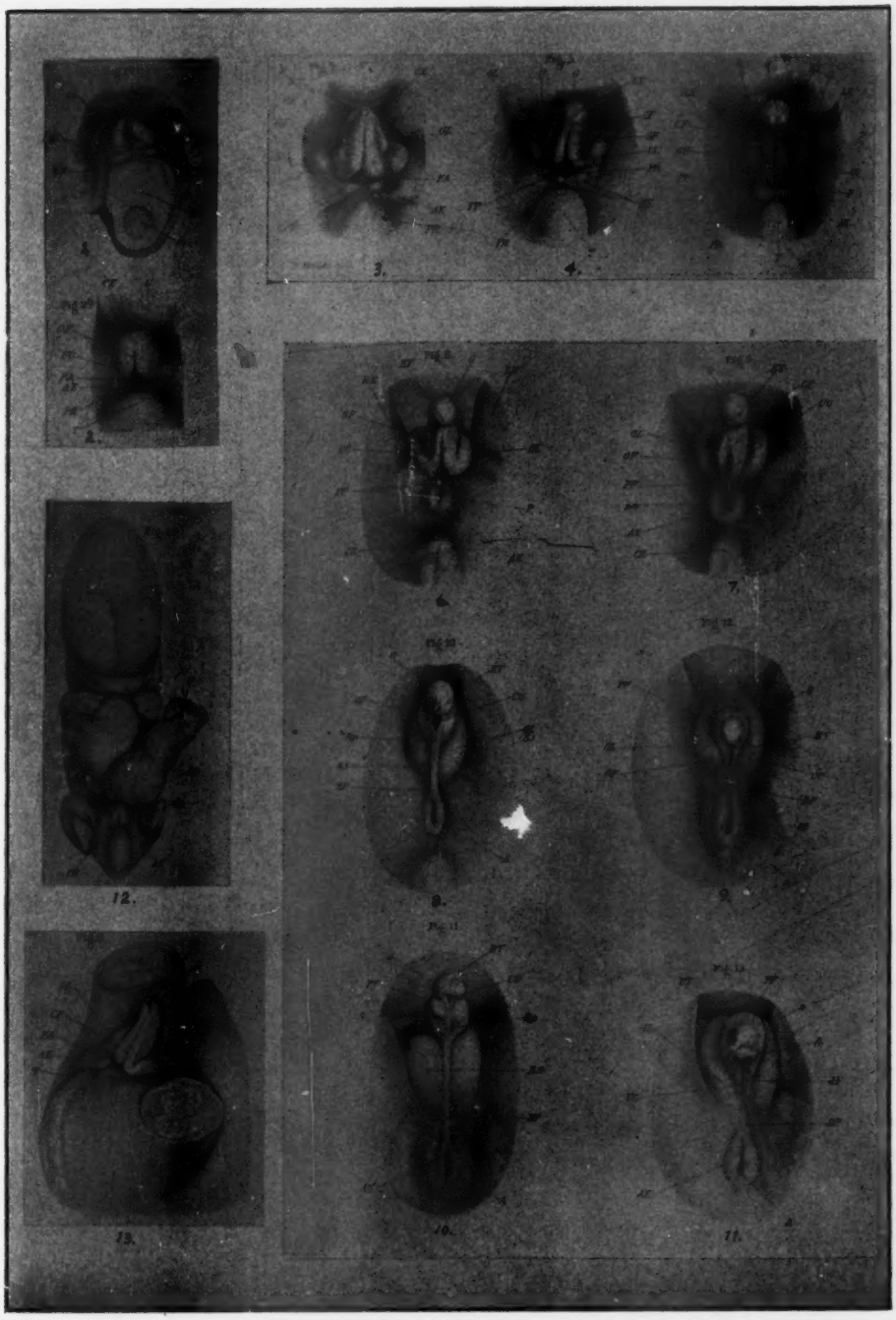


PLATE XV. 1, embryo, tail removed. Genital grooves on the under surface of the overhanging genital tubercle. 2, embryo of 19 mm., front view. 3, same embryo, genital region. 4, embryo of 21 mm., genital region. 5, embryo of 21 mm., genital region further developed. Somewhat older. 6, embryo of 23 mm., caudal end. 7, embryo of 26 mm., genital region. 8, embryo of 31 mm., male. 9, embryo of 39 mm., female. *pp*, area of fusion of the anal tubercles and genital folds. 10, embryo of 46 mm., male, genital region. 11, embryo of 58 mm., male, genital region. 12, embryo of 62 mm., female, genital region. 13, embryo of 82 mm., female. Early frenulum clitoridis. *A*, anus. *AE*, anal tubercle. *CE*, coccyx. *GF*, genital fossa. *CF*, tail filament. *CM*, cloacal membrane. *FA*, fissure ani. *FC*, frenulum clitoridis. *G*, glands. *GE*, genital tubercle. *GF*, genital fold. *LE*, left lower extremity. *P*, proctodeum or anal pit. *PA*, primitive anus. *PF*, preputial folds. *PP*, primitive midperineum. *PR*, precaudal recess. *RP*, raphé of perineum. *RP*, raphé of penis. *RS*, raphé of scrotum. *SU*, sinus urogenitalis. *S*, scrotum. *T*, tail. *U*, umbilical cord. *UC*, urogenital fossa. (After Otis.)

and remains attached to the levator and free from the rectal wall. This is the puborectalis muscle.

The downgrowth of the longitudinal muscle of the rectum receives fibers from the levator musculature and together they penetrate the musculature of the anal sphincter. The outer margin of the penetrating muscle lies at the border between the superficial and deep portions of the anal sphincter. The penetrating muscle composed of smooth and striated fibers invades the sphincter in a fan formation. Medially the fan penetrates the inferior portion of the internal circular coat of the gut. Thickening of the internal circular coat to form the internal sphincter does not occur until the penetrating fibers have acted on the circular bundles to cause them to override each other. The internal sphincter is formed by the action of its penetrating longitudinal musculature.

The portion of the external sphincter that lies free on the penetrating fan is the deep anal sphincter or from its relation to the longitudinal musculature the extrinsic sphincter externus ani. The portion of the sphincter within the penetrating fan is the superficial sphincter or, from its relation to the longitudinal musculature, the intrinsic external sphincter ani.

The smooth internal sphincter is formed by the longitudinal penetrating muscle and is intrinsic to the longitudinal wall.

External or Ectodermal Cloaca. The urogenital cloaca before its completion receives material from its functioning kidney and the intestinal tract. (The buccopharyngeal membrane has ruptured at the end of the third week.) The corresponding portion of the cloacal membrane yields to the pressure of its accumulating contents and forms small openings during the eighth week for its passage. The separation of the ventral from the dorsal cloacae is completed by the formation of the pelvic bar. Desquamation of the entodermal lining of the cloacal membrane leaves the thin sheet of ectoderm of the cloacal membrane with its multiple openings in the urogenital area which again drain the intestinal and urinary tracts. The passage —external cloaca—extends from the upper margin of the desquamated entodermal layer of the cloacal membrane through the anterior genital fossa to the outer margin of the genital folds. The urogenital cloacal membrane disappears and the urogenital sinus is open at the surface. A thick layer of cells derived from the edge of the anal cloacal membrane persists and forms a lining on the walls of the dorsal portion of the external cloaca. The pelvic bar by its dorsal surface forms the anterior wall of the forming anal canal.

Subsequently the perineal bar forms on the back of the pelvic bar. The anal canal is separated from the anterior genital tract. The thin ectodermal sheet separating the canal from the surface ruptures during the third month, under pressure of the intestinal contents above, and the anal canal opens to the exterior. The buccopharyngeal membrane has been open for a considerable time.

The derivation of the lining of the anal canal from the ectoderm to a small extent and the entoderm to a greater extent accounts for the transitional nature of the epithelial lining of the adult anal canal.

Development of Structures of the Pelvic Outlet. The mesoderm at the borders of the cloacal membrane proliferates under cover of the ectoderm and forms an elevated margin that surrounds the genital fossa whose roof is the submerged cloacal membrane. A broad conical eminence develops anteriorly, the genital tubercle, barely separated cranially from the umbilical cord. Its rounded free end as the primordium of the glans is faintly separated from the basal portion. Lateral slopes below the glans area extend into the outlying basal tissue as the lateral buttresses which will form the future corpora cavernosa. Caudally the slope extends to a horizontal bar on either side of the cloacal membrane connecting the anal tubercles behind the cloacal membrane. The anal tubercles grow forward and converge across the fossa to fuse in the midline on the posterior surface of the pelvic bar. The genital fossa is divided by the fused anal tubercles into a posterior anal pit and an anterior urogenital sinus.

The mesoderm at the margins of the urogenital sinus develops its structures in the mesoderm that helps form the infra-umbilical abdominal wall and becomes included in that portion of the abdominal wall.

Development of the Male Genitalia. The tubercle becomes elongated and forms lateral buttresses which migrate medially to form a somewhat cylindrical phallus whose base is separated from the surrounding body areas by the newly formed labioscrotal swellings; the latter appear first as broad elevated areas extending laterally from the base of the phallus, but are soon changed into swollen ridges separated from the phallus by the lateral phallic grooves. With the elongation of the tubercle to form the phallus, the urogenital opening becomes sharply outlined and the sex distinction becomes emphasized by the length of the opening. The genital folds are now the urethral folds. In the male the opening extends from the base to the apex of the phallus, whereas in the female it extends only

to the base of the glans. The lips of the urethral folds meet in the midline to form a raphé and transform the open urogenital sinus into the tubular urethra, open at its distal portion. The labioscrotal swellings take their final position caudal to the penis to form the scrotum, the halves of which becomes approximated to form the raphé in the midventral line and maintain their bilateral character. The glans develops the wide coronary sulcus and becomes sharply defined. The gradual closure of the urethral opening develops synchronously with the growth of the prepuce. The urethral opening shifts to a more terminal position in the frenular notch of the glans during the later stages of prepuce formation. The urethral opening becomes entirely closed and later there is formed the permanent terminal urethral opening at the apex of the glans.

The prepuce is first recognized as a pair of swellings on the inside of the urethral opening. These swellings gradually fuse together over the dorsum of the shaft to form a flat skin whose distal margin envelops the corona of the glans. This fold of skin grows distally until it covers the glans completely.

Development of the Female External Genitalia. The entire process of development of the external genitalia in the female is accompanied by fewer structural changes than occur in the male. Completion of development, however, takes a longer time so that final differentiation in the female does not synchronize with the more differentiated transformation in the male. The genital tubercle of the female resembles that of the male. The genital groove, however, in the female is shorter. The groove on the tubercle becomes obliterated and the genital folds separate from the groove. Characteristic of the female is the caudal decurvation which appears to be brought about by an excess in the growth of the cavernous over the urethral regions of the phallus. The urethral folds develop as caudal projections supporting the slightly overhanging glans, which becomes more clearly defined than in the male. The caudal ends of the labioscrotal swellings grow towards each other and join in the midventral line to form the posterior commissure. In this manner these originally paired swellings are transformed into a cranially opened horseshoe-shaped rim, enclosing the rest of the external genitalia and separating them from the anus. In the female the labioscrotal swellings form the labia majora. The shaft and glans of the phallus form the clitoris, and the ununited urethral folds separate from the clitoris and become the labia minora. The unobliterated urogenital orifice becomes the urethrovaginal orifice. The prepuce is formed as in the male but its growth is slower.

PART V

INTESTINAL CANAL—INTESTINAL LOOPS—ROTATION OF COLON—CECUM AND APPENDIX ATTACHMENTS OF MESOCOLON

Intestinal Canal. The diaphragm develops from the mesoderm of the head fold. The lateral portions developed from the closed pleuroperitoneal channels grow towards the median plane to fuse with the dorsal portions growing ventrally from the dorsal mesentery. On completion of the diaphragm, the abdominal portion of the cœlomic cavity is separated from the thorax. Incomplete fusion of the diaphragm usually on the left side leaves an opening through which a portion of the abdominal contents may pass into the thorax to form a congenital diaphragmatic hernia.

The peritoneal cavity is small. The dorsal wall of the embryo is strongly anteflexed, the space between the intestines and side walls is shallow and the greater part of its ventral aspect is taken up by the body stalk and vitelline duct. The peritoneal cavity is open to the extra-embryonic cavity.

The midgut forms a forward bend—enterocolic or first primary loop—which is attached at its apex to the vitelline duct. The primary loop is supplied by the superior mesenteric artery. The descending cranial limb of the loop is continuous with the terminal foregut whose developing stomach with its attached liver develop in the upper portion of the peritoneal cavity. The ascending caudal limb is continuous with the hindgut which terminates by expanding into the retroperitoneal entodermal cloaca. The two limbs lie side by side. The caudal limb develops a small expansion—cecum—which marks the beginning of the colon. The lengthened mesentery diminishing toward the mesogastrium above and terminating at the cloaca blow, is fan-shaped. The mesentery is attached along the middle of the dorsal wall between two elongated and rounded swellings—Wolffian bodies—which fill the dorsal wall between the mesentery and the lateral parietes.

The liver grows enormously and almost fills the peritoneal cavity. The growing intestinal tube is crowded into the extra-embryonic space between the vitelline duct and the median folds of the amniotic sac. The folds and ducts fuse by their outer coats of morula mesoderm beyond the projecting enterocolic loop and enclose a space—peritoneal space of umbilical cord—in which the ileal coils of the primary loop develop.

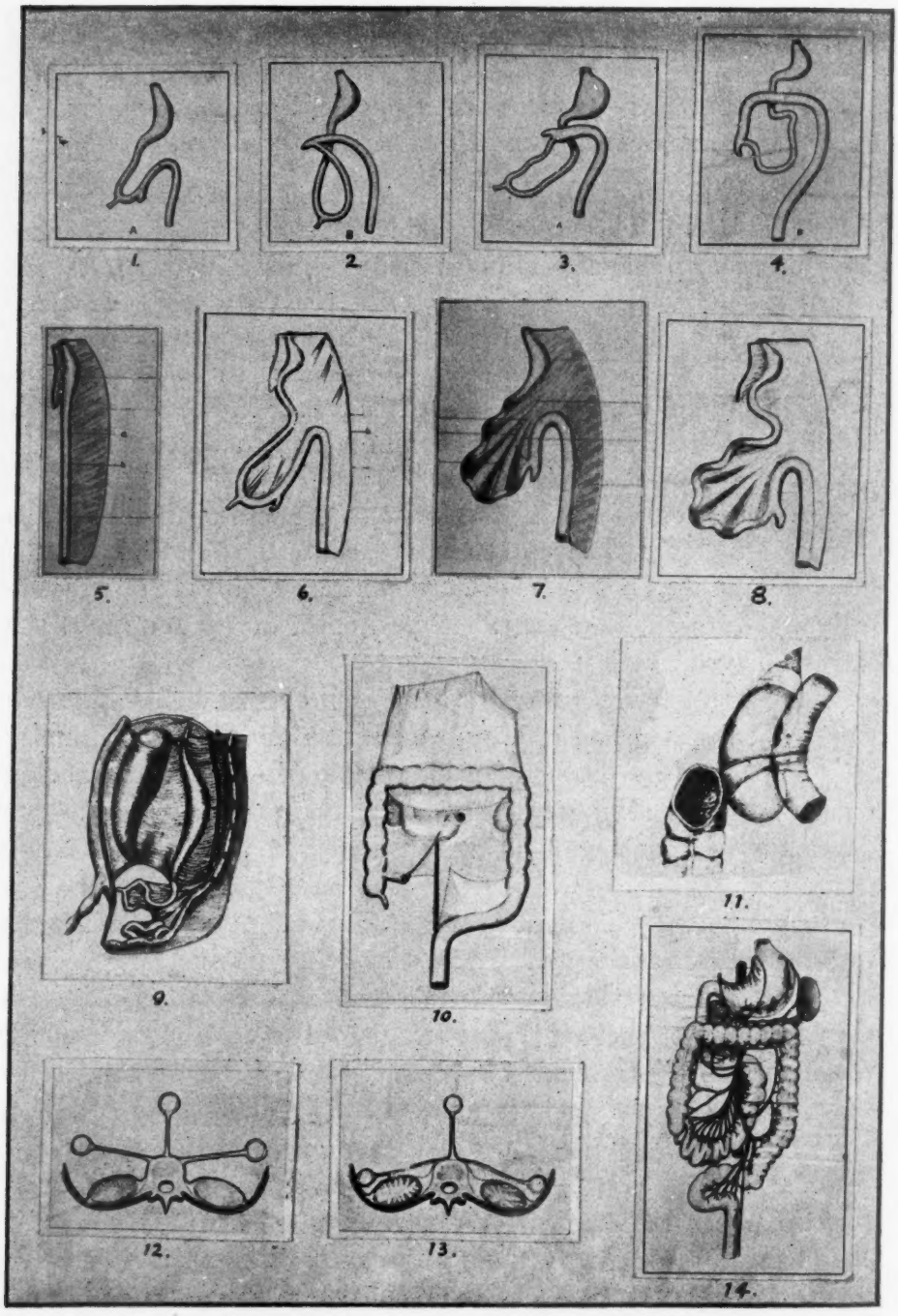


PLATE XVI. Rotation of intestinal canal. 1–4, rotation of intestinal tube. 5–8, early growth of intestinal loops and mesentery. 9, ventral and dorsal mesentery of gastrointestinal tract. 10, completed rotation of colon forming the infracolic compartment. Omentum turned up. Relation to kidneys, duodenum and superior mesenteric artery. Attachments of mesocolon shown by dotted lines. 11, right mesocolon in relation to right kidney and duodenum. 12, cross section of kidney and intestinal mesentery before fixation of lateral colon. 13, after fixation. 14, final arrangement of arteries of alimentary canal after rotation. (After Huntington.)

Second and Third Loops of the Three Primary Loops. The cranial limb of the enterocolic loop at its origin from the tapered swelling of the stomach bends laterally, with its convexity to the right abdominal wall and forms the gastroduodenal loop supplied chiefly by a branch of the celiac artery. The caudal limb of the enterocolic loop at its termination in the hindgut, bends laterally with its convexity toward the left abdominal wall and forms the left colic loop on the posterior abdominal wall, chiefly supplied by the inferior mesenteric artery.

The enterocolic loop in the peritoneal cavity of the cord with the intra-abdominal cephalic right gastroduodenal loop and caudal left colic loops are the fundamental loops which form the mesenteric portion of the intestinal canal. The enterocolic loop undergoes an elaborate development to form the distal part of the duodenum, the jejunum, ileum, cecum, ascending colon, and the right half of the transverse colon. The base of the enterocolic mesentery receives the superior mesenteric artery from the aorta at its center where the mesentery early becomes fixed to the ventral surface of the first lumbar vertebra. The left colic loop develops later than the first two fundamental loops to form the left half of the transverse colon, the splenic flexure, the descending colon, and sigmoid colon.

A portion of the mesocolon of the left colic loop under pressure of the neighboring intestinal coils early fuses with the dorsal peritoneal wall in the region of the left side of the left kidney and there fixes its attached gut.

The attachments of the mesentery to the region of the first lumbar vertebra by its enterocolic portion and to the left iliac fossa by its left colic portion are the most constant of the fixed points formed by the mesentery.

The rapid growth of the liver subsides, the ventroflexion of the embryo becomes a dorsoflexion at the lumbosacral angle, and the space of the left abdominal cavity enlarges. The vitelline duct, compressed by the median folds of the greatly distended amniotic sac, disintegrates and loses its attachment to the intestinal canal. The midgut, free in the extra-embryonic peritoneal cavity of the cord withdraws its coils into the roomier embryonic abdominal cavity where they complete their development.

The colic portion of the primary loop withdraws last from the extra-embryonic peritoneal cavity. The colic portion ascends ventrad to the surface of the intestinal coils and crosses the duodenum to bring the cecum into the right hypochondriac region below the pyloric end of the stomach and liver. The proximal portion

of the colon lengthens to form the ascending colon and bends toward the right iliac fossa where the cecum descends ventral to the kidney, and takes its adult position during the later prenatal or early postnatal period. The cecum may fail to descend and remain high in the abdominal cavity.

The rotation of the caudal ascending limb of the primary loop from a position below and to the left of the upper limb to a position above and to the right of the upper limb twists the mesentery of the loop around its axis of the superior mesenteric artery. A further rotation of the proximal ileocolic portion carries the ileal coils over the cecum and the ileum enters the cecum from the left side.

The colic portion of the twisted mesentery fuses with the posterior abdominal wall of the right side from the superior mesenteric artery on the duodenum to the cecum. Left of the artery the mesentery fuses with the ventral surface of the duodenum from which the line of fusion extends laterally to the anchorage of the descending colon. The voluminous intestinal coils crowd the ascending and descending colon laterally against the right and left lumbar walls. The apposed serous surfaces of the lateral colon, mesocolon and peritoneal wall fuse and the attached portions of the gut are retroperitoneal in the area of fusion. The retroperitoneal portion of the gut on its lateral surface is covered by an avascular sheet of peritoneum. The fused mesocolon helps form the prerenal peritoneum at each side.

The colon between its fixed regions in the left and right iliac fossae, stretches across the abdominal cavity as the transverse colon. The great omentum descends in front of the transverse colon and the apposed serous surface of the deep layer of the omentum fuses with the cephalic portion of the colon and mesocolon.

The colon in the left iliac fossa below its attached descending portion, bends to form a small loop—sigmoid loop—whose upper limb begins at the lower end of the descending colon and whose lower limb ends at the colon derived from the cloaca.

The sigmoid mesocolon partly retains its primitive vertical origin to the dorsal midline and partly extends horizontally to the fixed descending mesocolon. The vertical and horizontal lines of attachment meet at a right angle open inferiorly and to the left, to form the intersigmoid fossa. Deep to this angle of mesenterial attachment lies the left ureter.

The cecum first appears as a circumscribed thickening of the lower limb of the enterocolic loop. The thickening forms a conical pouch which becomes narrow at its apex where a narrow tubular extension of the appendix develops. The pouch of the cecum grows

beyond the apex and the appendix becomes a diverticular appendage. The three longitudinal muscular bands meet at the root of the appendix.

The colon, from the lateral fixation of its ascending and descending portion continues medially with the fixation of the cranial leaf of the transverse mesocolon to the omentum, forms the infracolic compartment of the posterior wall of the peritoneal cavity. The mesentery of the small intestines lies within the compartment.

PART VI

WALL OF COLON, RECTUM, AND ANAL CANAL—EPITHELIAL LINING, MUSCULAR WALL, GLANDS

Wall of Colon, Rectum, and Anal Canal. The colon or large intestine begins with the cecal swelling on the caudal limb of the enterocolic loop and ends with its attached dorsal mesentery at the expansion of the hindgut that forms the internal cloaca. The rectum, upon the division of the internal cloaca, begins at the termination of the mesenteric gut and ends on the entodermal or internal surface of the cloacal membrane. The anal canal begins at the entodermal surface of the cloacal membrane and ends at the inferior margin of the anal pit on the surface of the perineum to communicate with the exterior.

The changes that take place in the entodermal epithelial tube—mucous membrane—of the appendix, colon, and upper rectum are similar to those of the esophagus, stomach, and small intestines. The wall of the mesenteric colon, from within out, consists of the stratified epithelium surrounded by a zone of loose mesoderm bounded by a thin layer of forming muscle cells, the circular muscle wall; the circular layer is covered by a layer of mesoderm and the entodermomesodermal tube is invested by mesothelium excepting at the area of attachment of the mesentery.

Beginning in the upper rectum, the epithelium becomes thickened to form longitudinal ridges. The mesodermal wall indents the lumen and the ridges become folds of the entire wall. The process extends to the descending colon, and before extension to the left transverse colon occurs, a similar process begins at the ascending colon near the ileocolic valve. The transverse colon develops its folds last. The epithelium outside the folds develop conical projections which are the transitory villi of the colon. The sigmoid colon at the crest of its ridges develops goblet cells which are more numerous than in the descending colon later. At various points the folds fuse at their tops

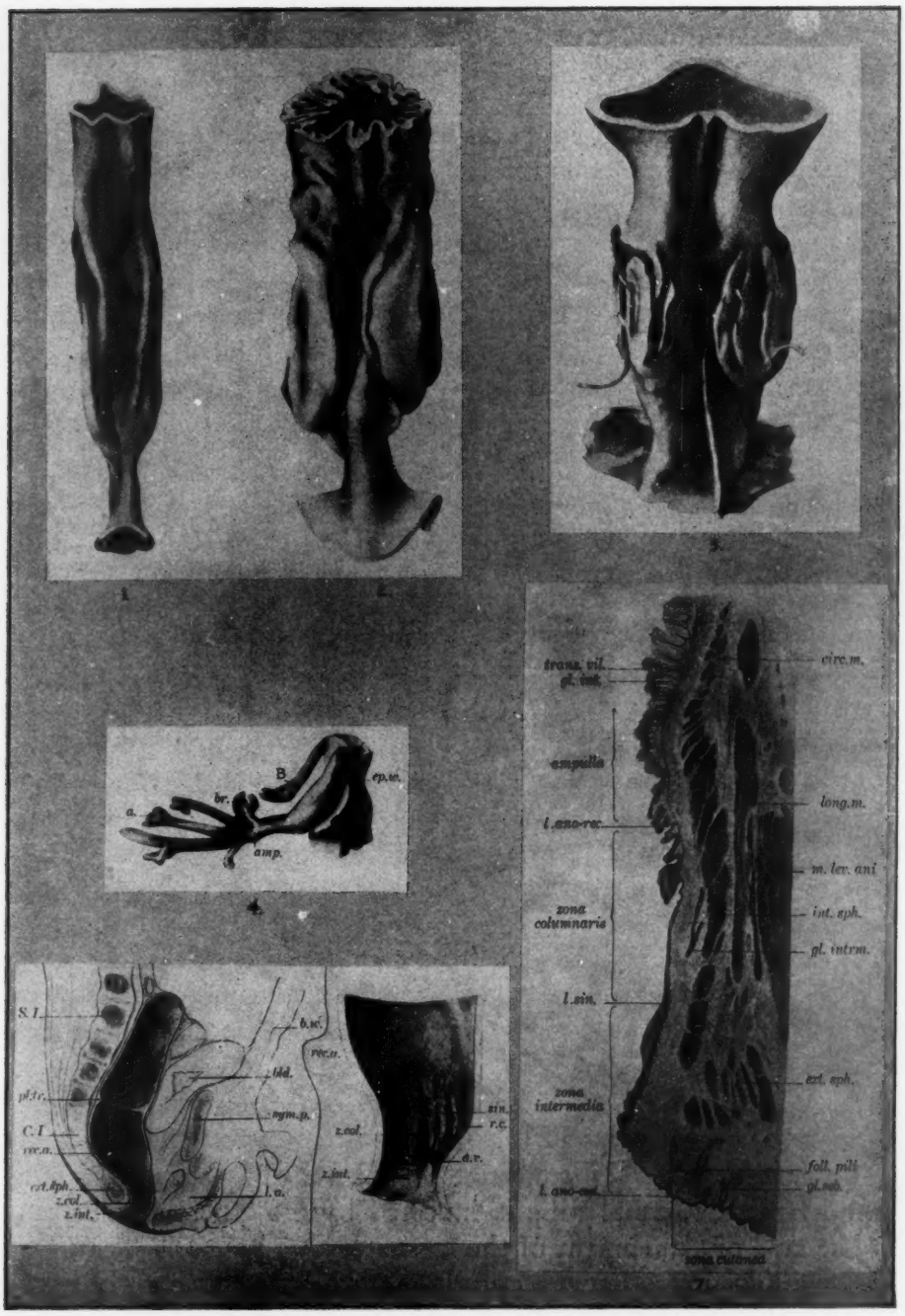


PLATE XVII. 1, 2, 3, reconstruction of epithelium of the pars analis recti of an embryo of 240 mm. Note in 3 slender extension from each side of epithelial tube is an intramuscular gland. 4, branched intramuscular gland in embryo of 245 mm. *a*, acinous termination. *br.*, branch. *amp.*, ampulla. *ep.w.*, epithelial wall. 5, sagittal section through pelvis at birth. *S.I.*, first sacral vertebra. *pl.tr.*, plica transversalis recti. *C.I.*, first coccygeal vertebra. *rec.a.*, rectal ampulla. *ext.spb.*, external sphincter muscle. *z.col.*, zona columnaris. *z.int.*, zona intermedia. *b.w.*, body wall. *bld.*, bladder. *sym.p.*, symphysis pubis. *l.a.*, levator ani muscle. 6, sagittal section through the lower part of the rectum. *rec.a.*, rectal ampulla. *z.col.*, zona columnaris. *z.int.*, zona intermedia. *sin.*, rectal sinus. *r.c.*, rectal column. *a.v.*, anal valve. 7, longitudinal section through the pars analis recti. *trans.vil.*, transitory villus of the ampulla. *gl.int.*, intestinal gland. *ampulla*, ampulla recti. *l.ano-rec.*, anorectal line. *l.sin.*, sinuous line. *l.ano-cut.*, anocutaneous line. *circ.m.*, and *long.m.*, circular and longitudinal layers of the muscularis. *m.lev.ani*, levator ani muscle. *int. spb.*, internal sphincter muscle of the anus. *ext.spb.*, external sphincter muscle of the anus. *gl.intrm.*, intramuscular gland. *foll.pili*, hair follicle. *gl.seb.*, sebaceous gland. (After Johnson.)

and form epithelial cysts. The epithelium degenerates and the cysts disappear particularly in the appendix and cecum. In the rectum, the cysts open to the lumen and become glands again.

The epithelium develops knobs of growth into the mesoderm which become the intestinal glands. The glands multiply by splitting longitudinally.

Under the distending pressure of the accumulating meconium the villi become flattened out and disappear; the mucosa becomes thinned out and the glands are spread apart and shortened.

Lower Rectum and Anal Canal: Anorectal Canal. The lower rectum, where it passes into the anal canal, develops ridges and folds which are transverse in direction. The division between the rectum and anal canal corresponds to the level of the pelvic floor. Early the rectal tube develops a spindle-shaped swelling which is elongated at first and then becomes more transverse at the bulbus analis and is the site of the rectal columns. Later, below the bulbus a second swelling appears—bulbus terminalis—limited inferiorly by the anal membrane, and is the site of the future zona intermedia.

The bulbus analis develops three longitudinal folds which extend throughout its length and end below on the zona intermedia. The posterior and lateral portions are each folded. Secondary folds develop in considerable numbers and obscure the primary folds. The larger infoldings of the mucosa form the rectal columns—columns of Morgagni—and are present in great numbers. Sixty were counted by Johnson in a 240 mm. embryo. The smaller folds are similar in structure to the more conspicuous columns. The columns, at their inferior portion, are joined together by a tag—anal valve—and the depression between the raised columns—rectal sinus—continues below their union to form the recess of the crypt.

Early the pars analis is lined by a few layers of low columnar epithelium similar to the epithelium of the early digestive tract. This zone of epithelium marks the transition of the mucosa of the intestinal tract above to the skin of the surface below. This transition passes through two superimposed areas to reach the skin margin—anocutaneous border—the zona columnaris, and the zona intermedia. The simple columnar epithelium becomes the stratified columnar epithelium of the zona columnaris. The stratification consists of two to three layers, and extends above for a short distance on the rectum to form the anorectal line. Below, the stratification extends at various points into the anal skin folds which extend inward and upwards from below.

The zona columnaris, at its rectal sinuses, develops tubular glands which lie in the submucosa. Six or eight of the simple tubular

glands become branched and penetrate the neighboring muscular wall to form the intramuscular glands. The main ducts of the gland extend outward and usually downwards, and penetrate the internal sphincter muscle where a swelling of the duct gives off several tubular branches that end blindly in the intramuscular connective tissue. Occasionally a tubule penetrates the outer longitudinal muscle. An occasional tubule forms acini. A small amount of lymphoid tissue forms around the terminations of the tubules. Stratified cuboidal epithelium is found in the larger ducts of the intramuscular glands, and simple cuboidal epithelium in the branches of the duct.

The intramuscular glands show evidence of secretory activity in the fetus and at birth.

The zona intermedia is lined by stratified squamous epithelium composed of several layers of the polygonal cells forming a thicker layer than the cells of the epidermis. Dermal papillae are present but hairs and sweat glands are absent. In the lower part of this zone are occasional sebaceous glands without hairs, and the epithelium is slightly cornified. The transition of the epithelium from the zona columnaris above is abrupt. The change in epithelium occurs at the level of the anal valves from which the changing level extends upwards on the summits of the rectal columns. The abrupt transition takes place along a zigzag line—anocutaneous line of Herrmann, or linea sinuosa analis. The zona intermedia, below, gradually goes over into the skin, and forms with the skin the anocutaneous line which is marked in the skin by the first hair follicles. The anocutaneous line is not well marked. The skin of the immediate neighborhood of the anus is the zona cutanea and is characterized by large sweat glands—the circumanal glands of Gay.

The muscularis mucosae of the rectum is similar to the colon above. Early, the muscularis mucosae extends into the zona columnaris as a thin sheet of fibers lying close to the epithelium. Later, fibers of striated and smooth muscle appear in the submucosa. The striated fibers of the rectal wall are terminal fibers of the rectal levator muscle.

The submucosa becomes fibrous. Numerous nerve fibers, blood vessels and Paccinian corpuscles are present in the submucosa.

The internal circular and outer longitudinal muscular coats of the colon continue downward to form complete muscular coats of the rectum. The inner circular muscle ends abruptly below. The outer longitudinal muscle, continuous around the gut, becomes thickened into bands at the antimesenteric and lateral borders to form the teniae coli. The teniae extend from the base of the appendix to the

end of the mesentery at the beginning of rectum. The outer longitudinal coat completely invests the rectum and by its external surface is attached in front to the pelvic bar, laterally and posteriorly to the levator musculature, and posteriorly at its in inferior termination to the rectococcygeus muscle. Above the longitudinal coat of the rectum is covered on its anterior and lateral surfaces by peritoneum, middle portion only anteriorly, lower portion extraperitoneally, the posterior surface is entirely extraperitoneal. The posterior surface of the rectal tube separates from the anterior surface of the sacrum when the longitudinal muscle is formed there. The separation of the gut from the sacrum leaves a space—retrorectal space—between them. The retrorectal space is bounded inferiorly by the attachment of the longitudinal muscle to the rectococcygeus muscle from the lowest piece of the sacrum. Above, the space is bounded by the lower branches of the inferior mesenteric artery. The outer longitudinal muscle of the rectum with fibers from its attached levator muscle continues beyond the internal circular coat below and spreads out in a fan. The muscular fibers of the fan end in small connective tissue fibers which penetrate the smooth internal circular muscle and the external sphincter muscle; and as septae of the penetrated musculature divide them into bundles. The septae attach to the lining of the anal canal. The internal circular and the external sphincter bundles are attached to the angles of the fan and enclosed within the space formed by the limbs of the longitudinal muscle and the covering deep surface of the anal lining. The internal circular muscle attached to the penetrating longitudinal muscle forms the internal or smooth sphincter. The internal circular fibers extend to the constriction between the bulbus analis and bulbus terminalis, and later when the bulbus terminalis disappears, extends into the region of the zona intermedia. The external sphincter surrounds the middle part of the zona intermedia.

The rectum develops infoldings of its mucous membrane to form transverse rectal folds. The folds become more pronounced and with further growth of the rectum become spread apart.

Early, all of the pars analis recti lies between the third sacral vertebra and the tip of the coccyx. At birth the pars analis lies below the tip of the coccyx.

Upon the completion of the development of the anal canal, external genitalia, and infra-umbilical abdominal wall from the mesodermal borders of the cloacal membrane and the band on the under surface of the umbilical cord, the developed structures preserve evidence of their common origin in the form of attachments to each

other. Similarly, the division of the internal cloaca into rectal and urogenital portions with the formation of the pelvic cavity preserves evidence of their common origin. The pelvic cavity is in relation to the attachments formed from the internal cloaca and the external cloaca. The urogenital and the rectal divisions remain joined by the pelvic bar at the inferior boundary of the pelvic cavity. The pelvic bar is contiguous by its external surface with the region of the fused anal tubercles which forms the continuation of the anal musculature into the urogenital musculature. The urogenital musculature with its attached urogenital organs develops in association with the lowermost portion of the infra-umbilical abdominal wall. The wall with the deep portion of the urogenital sphincter cloacae, forms a space within which are confined the structures of the urogenital sinus. The space is the superficial perineal pouch.

Muscle is a differentiated product of mesoderm whose less differentiated portion forms the connective tissue envelope for the muscle fiber. Degeneration of muscle forms aponeurosis, fascia, and tendon. The final form of a muscle fiber, striated or unstriated, is the resultant of tensional stresses.

More Common Malformations of the Anorectal Canal. Usually the intestinal tract opens on the inferior surface of the embryo by a continuous canal. Infrequently, the lower passage is discontinuous or communicates with the nearby urogenital passage.

Failure of the pelvic bar to fuse with the cloacal membrane leaves a fistulous communication between the rectum and bladder. Invasion of the entodermal wall of the rectum by mesoderm obliterates the lumen and reduces the entodermal tube to a ligament.

Incomplete union of the anal tubercles leaving a shallow groove between them, followed by union over the groove by the labioscrotal folds, results in a fistulous opening in the anal canal that communicates with the vestibule of the vagina or penile urethra.

The cloacal membrane invaded by mesoderm forms a persistent fibrous membrane blocking the outlet of the anal canal.

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